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Original Communications

PULMONARY AND BLOOD GAS STUDIES IN EXPERIMENTAL ASPHYXIA AND ASPHYXIAL (ANOXIA) RESUSCITATION

COMPARISON OF METHODS OF RESUSCITATION

GEORGE L. BIRNBAUM, M.D.,* AND SAMUEL A. THOMPSON, M.D., F.A.C.S.
WITH THE TECHNICAL COLLABORATION OF EUGENE OSTROW
NEW YORK, N. Y.

THERE are many factors in the success or failure of resuscitation procedures. Of these factors which may determine the outcome, we have been impressed by the importance of two, (1) the prompt institution of resuscitation procedures and (2) the inherent effectiveness of the procedures. It is important to keep in mind that circulation and respiration go hand in hand and are interdependent; therefore, resuscitation procedures may have to be directed from the viewpoint of an adequate supply of oxygen to the lungs and an adequate circulation to carry oxygen to the heart muscle and brain as quickly and efficiently as possible.

In the minds of the laity and the profession there is still the belief that manual artificial respiration is the procedure of choice in asphyxia.

Lougheed, Janes, and Hall,[9] in an excellent experimental investigation, came to the conclusion that,

> As a result of our experiments we consider that prompt, adequate, and prolonged artificial respiration is the fundamental treatment for drowned, asphyxiated, or electrocuted persons. In the case of drown-

*Major, Medical Corps, U. S. Army.
From the Laboratory of Surgical Research and the Departments of Surgery and Thoracic Surgery, New York Medical College, Flower-Fifth Avenue Hospitals.
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> ing the additional procedures as outlined above are recommended for the reasons stated (p. 426).
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> Asphyxia may be maintained for only a comparatively short time if recovery is to take place. This time varied with the individual animal but rarely exceeded four minutes; in some animals recovery has occurred after the trachea had been clamped for seven minutes, while other animals failed to recover after temporary asphyxia of two minutes duration. Another interesting feature was observed in the time interval between the cessation of respiratory efforts during asphyxia and the cessation of expulsive cardiac beats. This interval varied only from eleven to seventeen seconds. If the asphyxia was maintained four or five seconds longer, resuscitation was of no avail.
>
> . . . it must be emphasized, however, that successful artificial respiration was possible only when started within a few seconds after the time when spontaneous recovery could take place. Precisely the same criteria had to be applied in the cases where asphyxia was produced by drowning.

Henderson and Turner[7] believe that manual artificial respiration is more effective than mechanical suck-and-blow resuscitation. However, they have found that the rhythmic suck-and-blow mechanism, using pressures of plus 15 and minus 15 mm. Hg, could do no serious harm to the lung. Coryllos[8] was of the opinion that the suck-and-blow resuscitator was superior to manual artificial respiration.

In view of the conflicting opinions on methods of resuscitation, we carried out a group of experiments to see if some definite conclusions could be drawn. This communication deals with (A) obstructive asphyxia and inhalation asphyxia with nitrogen and (B) pulmonary and blood gas studies at various stages of asphyxia. Drowning is essentially a form of obstructive asphyxia,[8, 9] both as to mechanism and treatment. It requires no additional consideration other than measures to overcome spasm of the larynx and to clear the airways of fluid.

TECHNIQUE

For detailed technique the reader is referred to previous communications. When asphyxia was produced by inhalation of inert gas, there was an underwater valve for "blowing off" the exhaled air in order to prevent rebreathing of carbon dioxide. Dogs weighing 8 to 12 kg. were anesthetized by the intraperitoneal injection of veterinary nembutal (sodium pentobarbital; sodium ethyl, 1 methyl-butyl, barbiturate) 1 c.c. (1 gr.) per 5 pounds. Obstructive asphyxia was accomplished after the application of a special fitting face mask. In other experiments asphyxia was carried out by introducing and clamping an intratracheal rubber tube with inflatable rubber cuff to make the trachea leakproof.

In the pulmonary and blood gas experiments, intratracheal tubes were used for convenience. Obstructive asphyxia was carried out by clamping the tube. Inhalation of gas or resuscitation was carried out through the intratracheal tube. Pulmonary gas samples were obtained

by means of a 4 mm. rubber tube which extended in a leakproof manner through the intratracheal tube down as far as it would go into the lower lobe of the bronchus. Analysis of blood for oxygen and total carbon dioxide was made, using the Van Slyke manometric analyzer. Analysis of the pulmonary gas was made with the Haldane-Henderson-Bailey gas analyzer.

For plotting gas values, the duration of phases was taken from Fig. 1 as a standard. The gas values of other experiments were fitted in on this basis in their appropriate fraction or portion of each standard phase. For example, when a lung or blood sample was taken at the first third, second third, or last third of a particular phase of a particular experiment, the oxygen and carbon dioxide values would be plotted in the appropriate third of the standardized graph phases.

DISCUSSION

A. *Comparison of Methods of Resuscitation* (Figs. 9 to 13).—Table I reveals that manual artificial respiration with oxygen inhalation is the least effective of the methods tested. Rhythmic inflation (plus 14 mm. Hg) and suction (minus 9 mm. Hg), using oxygen, was by far the most effective of the methods and could be used at much longer periods after cessation of respiration. Table II shows the value of the suck-and-blow mechanism in anoxic resuscitation with nitrogen (asphyxial resuscitation). We have shown that such resuscitation is largely a reflex mechanism from the lung in which the vagus and carotid sinus

TABLE I

EFFICIENCY OF DIFFERENT RESUSCITATIVE PROCEDURES
OXYGEN

	SUCCESS	FAILURE	PERCENTAGE OF SUCCESS
Manual artificial respiration	6	5	55
Rhythmic inflation	7	2	78
Rhythmic suction	4	1	80
Resuscitation (rhythmic inflation and suction)	21	1	95

TABLE II

NITROGEN*

	SUCCESS	FAILURE	PERCENTAGE OF SUCCESS
Manual artificial respiration	1	6	15
Rhythmic inflation	2	10	17
Rhythmic suction	1	4	20
Resuscitation (rhythmic inflation and suction)	30	5	85

*Our analyses of the commercial nitrogen used showed no oxygen content, using the Haldane-Henderson-Bailey gas analyzer. According to the chemists of the commercial gas company whose nitrogen we used, the oxygen contamination is not over .02 per cent. In other words, the nitrogen was at least 99.98 per cent pure.

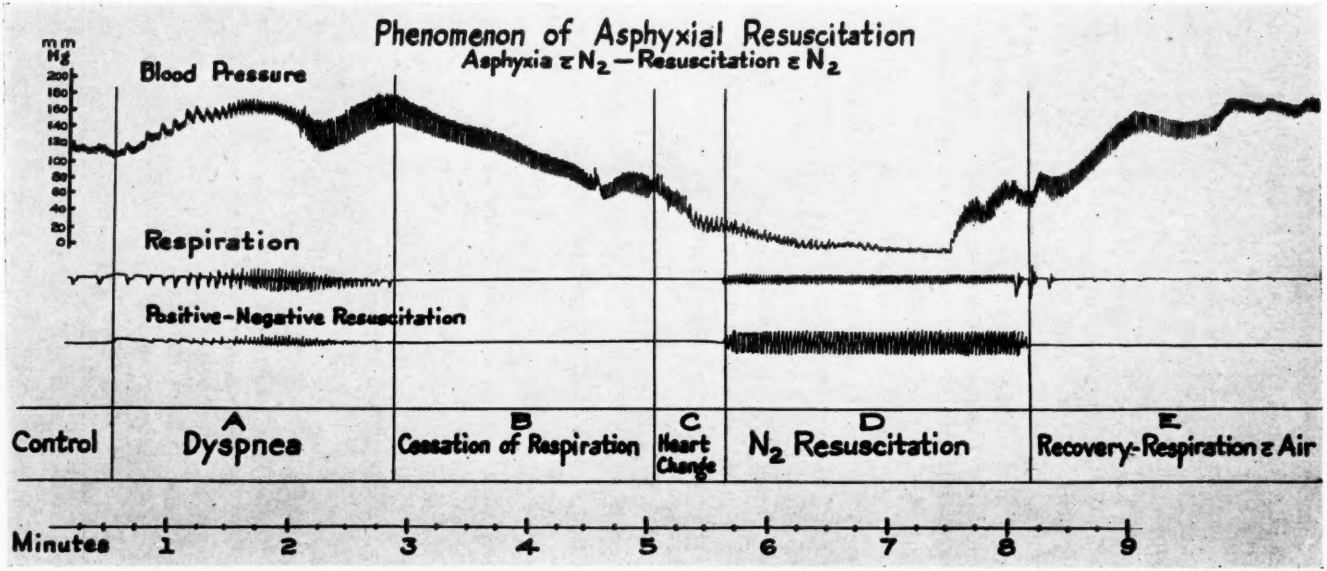

Fig. 1. (Dog 43).—The phenomenon of asphyxial or anoxic resuscitation. This illustration was used as a standard for plotting the gas values in the periods of asphyxia, as shown in Figs. 2 to 7. The gas values of Figs. 2 to 7 were placed in their appropriate fraction or portion of each standard phase. For example, when a lung or blood gas sample was taken at the first third, second third, or last third of a particular phase of a particular experiment, the oxygen and carbon dioxide values would be plotted in the appropriate third of the standardized graph phases. Notice that in period D (rhythmic pressure and suction on the lung with nitrogen) the circulation recovers before spontaneous respiration starts. (Courtesy of Surg., Gynec. & Obst.)

are concerned.[6] It is, probably, partly the superior and regular ventilatory excursion of the lung which accounts for superiority of one method over another.

Of course, we do not wish to imply that inert gas should be used clinically; it is evident that oxygen, or carbogen, should be used. Nor do we wish to give the impression that manual artificial respiration is not a valuable procedure. On the contrary, manual artificial respiration is still probably the most generally available procedure to combat asphyxia. It should be immediately instituted when respiration has ceased and continued until respiration returns, or until other and more efficient facilities are available. Likewise, rhythmic inflation of the lung, by mouth-to-mouth inflation, may be a valuable emergency procedure.

Rhythmic suck-and-blow resuscitation gives a more effective pulmonary ventilation than manual artificial respiration, or rhythmic inflation. This is due, for one reason, to the fact that suck-and-blow resuscitation does not depend upon muscle tonus of the respiratory muscles (as do manual artificial respiration, rhythmic inflation alone, or rhythmic suction alone) for adequate pulmonary ventilation. Acarbia (carbon dioxide deprivation), if feared, may be handled by the addition of 5 to 7 per cent carbon dioxide to oxygen.

It goes without saying that when respiration still is present in asphyxia, or where mechanical resuscitation begins to act "out of step" with the patient's respiration (signalling the return of spontaneous respiration), mechanical resuscitation should be discontinued and simple inhalation of oxygen, or carbogen, substituted for it).

B. *Pulmonary* and Blood Gas Studies at Various Stages of Asphyxia* (Figs. 2 to 7).—We have shown that asphyxial (anoxic) resuscitation (Fig. 1) is largely a reflex mechanism.[6] However, it requires more than reflex impulses to keep heart, muscle, and brain centers alive without oxygen. It was our first impression that such resuscitation might be due to a mobilization of oxygen from stagnating blood in vessels, or capillary pools, or even tissues. Blood gas analyses, however, have shown that such mobilization could play only a minor part, if any. In fact, when the respiration has ceased in asphyxia, the arterial and venous blood oxygen content is already almost at the base line; that is, not over 5 to 9 per cent of its normal content of 20 volumes per cent which is equal to less than 1 to 2 volumes per cent. Moreover, during the application of anoxic resuscitation, the blood oxygen level varies but slightly, remaining almost at the base line.

Theoretically, even if all the oxygen in the total blood volume could be utilized at this stage of asphyxia, it could not account for the suc-

*In Figs. 4 and 7 it is evident that at the end of period A or start of period B, when respiration has just ceased, pulmonary O_2 and CO_2 are about 8 and 6 per cent, respectively, for obstructive, and 2 and 4 per cent for N_2 asphyxia. This is somewhat in keeping with blood O_2 and CO_2 values for the same point on the graphs but does not indicate complete equilibrium between blood and alveolar gases. As period D is entered, the low gas values of the blood and lung run parallel.

cess of asphyxial, or anoxic, resuscitation. An adult human being requires about 300 c.c. of oxygen per minute under basal conditions. A dog of 10 kg. would require about 50 c.e. of oxygen per minute. If

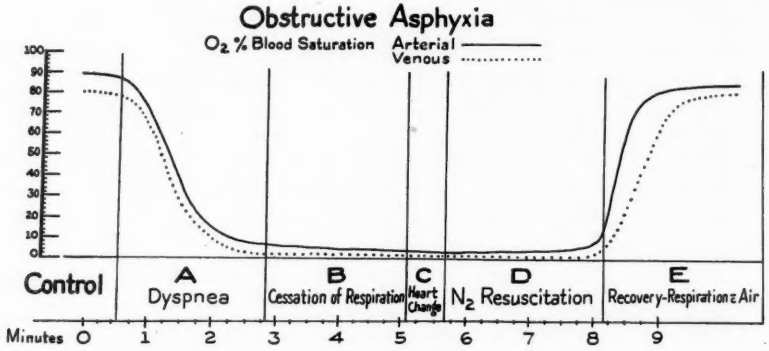

Fig. 2.—Oxygen saturation of the blood, in per cent, for the various periods of obstructive asphyxia (composite graph for Dogs 50, 56, 57, and 60). Courtesy of Surg., Gynec. & Obst.)

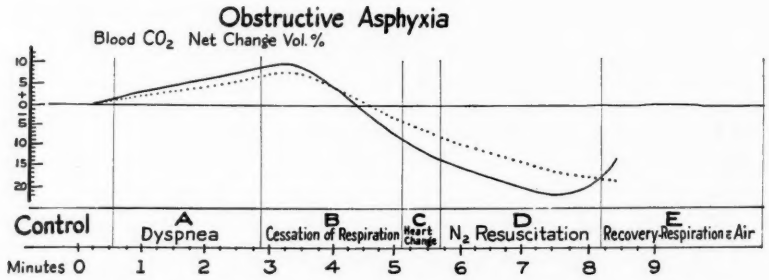

Fig. 3.—Total carbon dioxide content of the blood, in volumes per cent net change, for the various periods of obstructive asphyxia (composite graph).

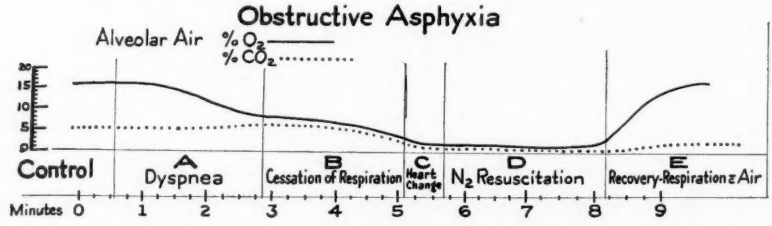

Fig. 4.—Pulmonary oxygen and carbon dioxide, in volumes per cent, for the various periods of obstructive asphyxia (composite graph).

the animal had even 900 c.c. of blood, then 2 per cent of this would equal 18 c.c. of oxygen in the blood, or only about enough for one-third of a minute. We already know that life can be maintained by anoxic resuscitation for much longer periods than this (Fig. 8).

That is to say, if, instead of discontinuing suck-and-blow resuscitation with nitrogen after the circulation and respiration has been restored, one persists in its use, the blood pressure oscillates and respiration is irregular; but life can be maintained for several minutes (in

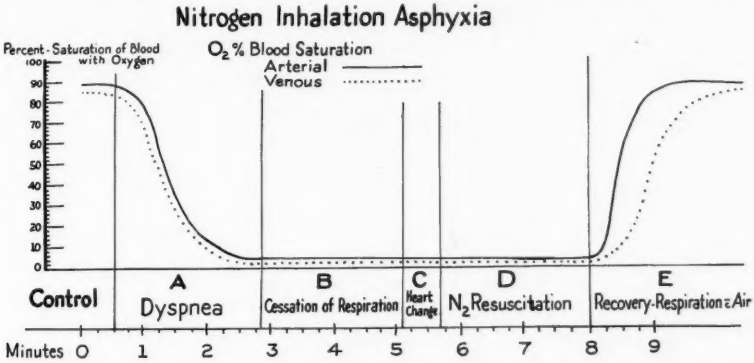

Fig. 5.—Oxygen saturation of the blood, in per cent, for the various periods of nitrogen inhalation asphyxia (composite graph for Dogs 51, 52, and 55). (Courtesy of Surg., Gynec. & Obst.)

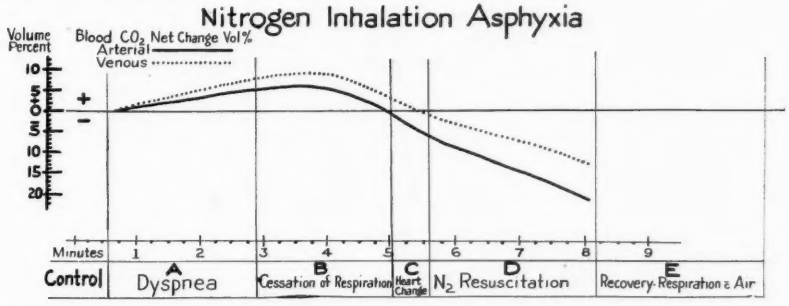

Fig. 6.—Total carbon dioxide content of the blood, in volumes per cent net change, for the various periods of nitrogen inhalation asphyxia (composite graph).

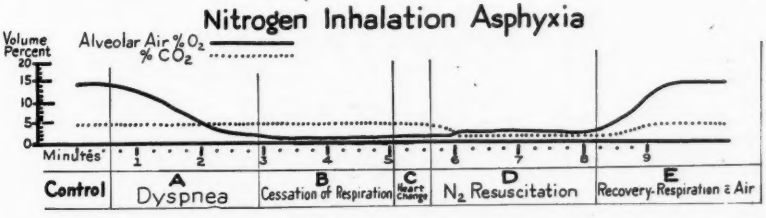

Fig. 7.—Pulmonary oxygen and carbon dioxide, in volumes per cent, for the various periods of nitrogen inhalation asphyxia (composite graph).

some cases for seven minutes or more) in this way. *What is the source of energy for the heart and brain centers?* To answer this we must turn to the question of blood carbon dioxide values at various stages of asphyxia and the factors in the acid base control of the body.

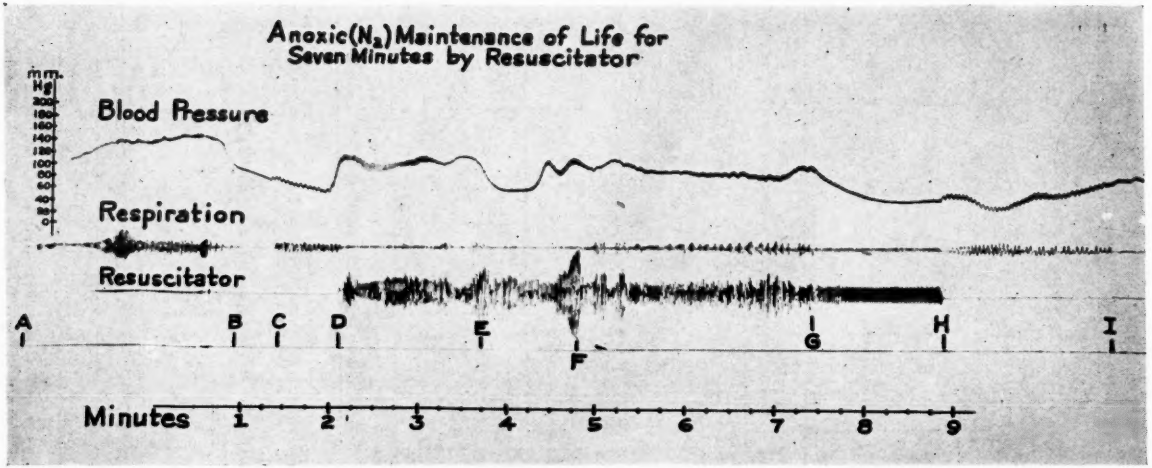

Fig. 8.—Anoxic (N_2) maintenance of life for seven minutes by suck-and-blow resuscitation with nitrogen (rhythmic inflation to plus 14 mm. Hg alternating with rhythmic suction to minus 9 mm. Hg). Nitrogen inhalation asphyxia by way of a face mask (Dog 5). *A*, control period after which asphyxia was started; *B*, respiration has ceased; *C* to *D*, manual artificial respiration with nitrogen inhalation unsuccessful; *D* to *H*, a period of seven minutes during which the resuscitator with nitrogen maintained life. Points *E* and *F* are two of many similar ones where the augmented height and depth of the resuscitator curve indicates respiratory stimulation as evidenced by the animal's resistance to pulmonary inflation and deflation by the resuscitator. Contrast this with period *G* to *H*, where the uniform height of the resuscitator curve may indicate final fatigue of the respiratory mechanism. *H*, resuscitator discontinued and manual artificial respiration with oxygen inhalation continued to point *I*.

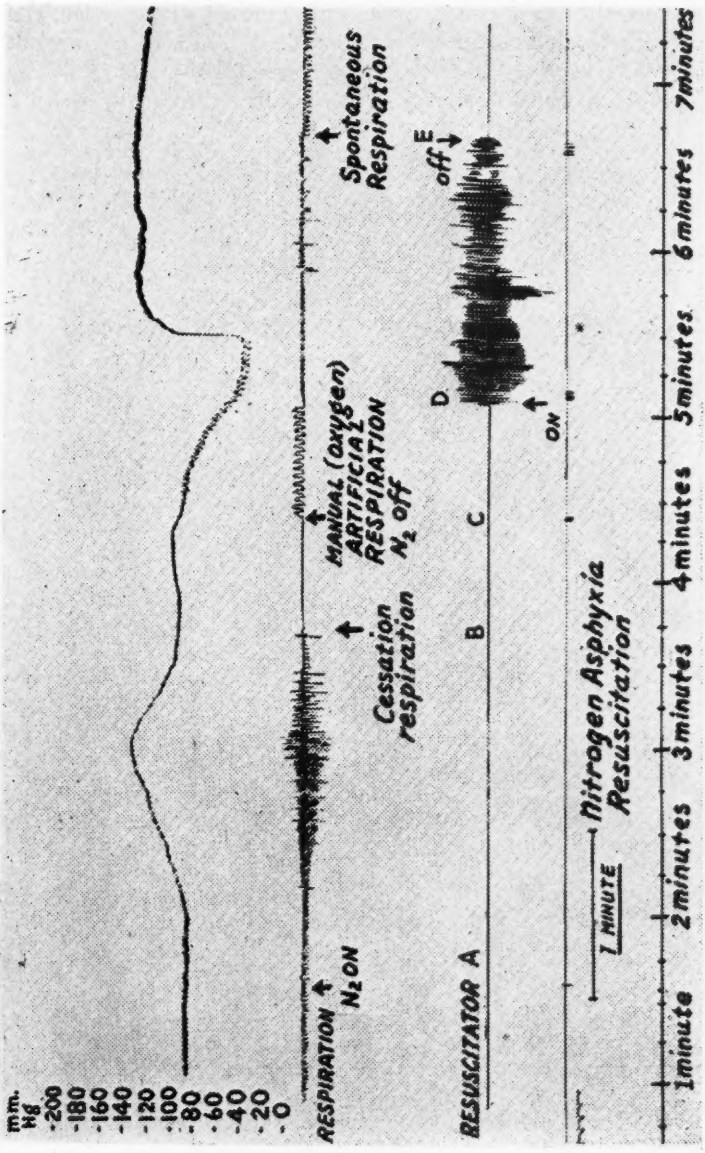

Fig. 9 (Dog 6).—Nitrogen inhalation asphyxia by face mask. *A*, Asphyxia started; *B*, respiration ceases; *C* to *D*, manual artificial respiration with oxygen inhalation fails. *D*, Resuscitator with oxygen is started and is followed shortly by resuscitation. (Courtesy of J. A. M. A.)

We have divided asphyxia in our experiments into the following stages: control, dyspnea, cessation of respiration, heart change preceding failure of the circulation, asphyxial (anoxic) resuscitation, and recovery after resuscitation is discontinued. It has been pointed out elsewhere that a period of initial apnea preceding dyspnea is usually described. It has not been marked or else was absent in our experiments.

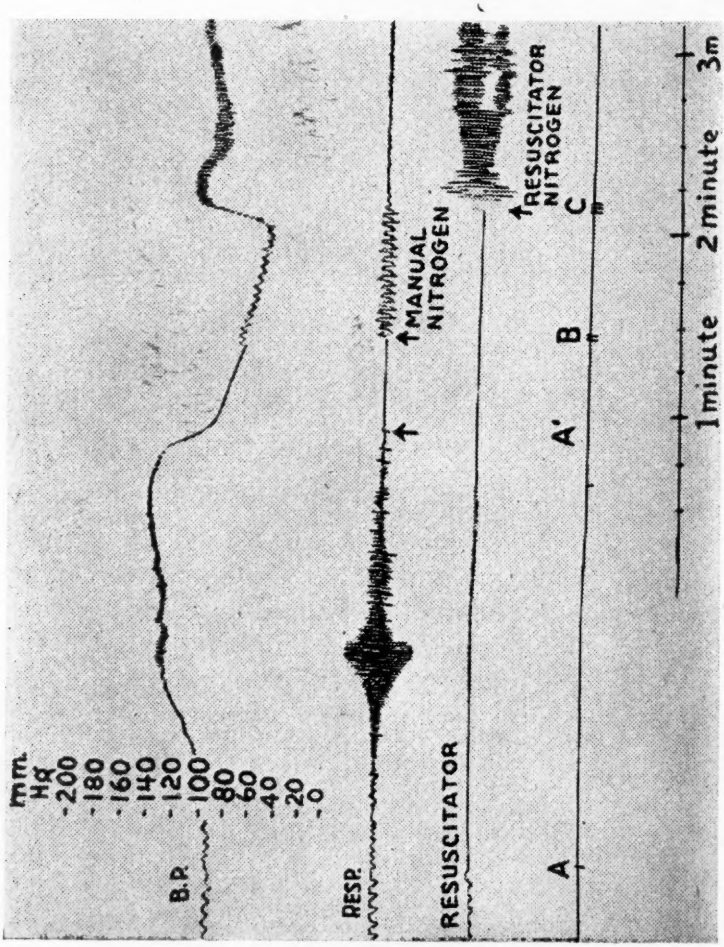

Fig. 10 (Dog 5).—Nitrogen inhalation asphyxia by way of face mask. *A*, Asphyxia was started. At *A'* respiration has ceased. *B* to *C*, manual artificial respiration with nitrogen inhalation fails. *C*, Resuscitator with nitrogen causes a prompt rise of blood pressure and resuscitation. (Courtesy of J. A. M. A.)

In the phase of dyspnea, the total CO_2 content of the blood rises. This rise continues into the first part of the period when respiration has ceased; the CO_2 begins to decline at about the first third or half of this period and continues to decline during the period of anoxic resuscitation.

Besides blood proteins and disodium-hydrogen-phosphate, bicarbonates are important buffers of the body.* It is known that 100 c.c. of blood have approximately 45 c.c. of total CO_2 of which (a) 2 to 3 c.c. are held in physical solution in the plasma, (b) 20 c.c. are in the plasma as bicarbonates, and (c) 20 c.c. are combined with plasma protein or hemoglobin. It is seen that the volume of CO_2 that can be held in physical solution is relatively small.

Hemoglobin is acid in reaction and can combine with the basic elements, sodium or potassium; these bases in turn attract CO_2. Oxyhemoglobin is even more acid than reduced hemoglobin and gives up its CO_2 more readily. Thus,

$$\text{in the tissues: } NaHb + CO_2 = NaHbCO_2$$
$$\text{in the lungs: } NaHbCO_2 + O_2 = NaHbO_2 + CO_2$$

In the maintenance of the constancy of the hydrogen-ion content of the body, the ratio of nongaseous blood CO_2 to gaseous CO_2 is a constant:

$$\frac{NaHCO_3}{H_2CO_3} = K = \frac{20}{1}$$

In extreme alkalosis, the nongaseous CO_2 is increased, and this is reflected in an increased alveolar CO_2 tension. In acidosis, the nongaseous CO_2 is decreased and this is reflected in a diminished alveolar CO_2 tension. In other words, an increased total blood CO_2 content usually means alkalosis; a decreased total blood CO_2 means acidosis. For practical purposes, the only other condition which could reduce the total CO_2 content of the blood without acidosis would be hyperventilation washing CO_2 out of the blood; and this could certainly not be a factor during the period of cessation of respiration in asphyxia. Likewise, there is one condition which could increase the total CO_2 content of the blood without indicating alkalosis, namely, gaseous acidosis from asphyxia or breathing atmospheres high in CO_2. This is represented as follows:

$$NaCl + H_2CO_3 = HCl + NaHCO_3$$

The bicarbonate stays in the plasma. The HCl is partly neutralized by plasma proteins, but the remainder combines with potassium in the red blood cells (Hamburger's phenomenon).

We shall mention two more factors important in the acid base regulation, since these may be related to our CO_2 findings and the possibility of anaerobic metabolism.[1, 10] The first is the role of the liver which conjugates and neutralizes acid metabolites; deglycogenation or

*The total alkali reserve of the body is considered equivalent to neutralize 1 l. of one-tenth normal hydrochloric acid; it is represented as follows:

a. 30 per cent in the interstitial tissues
b. 13 per cent in the hemoglobin
c. 40 per cent in the tissues
d. 17 per cent in the plasma

Acidosis may be associated with dehydration. Alkalosis may be associated with dehydration, edema, renal failure, or tetany. (D. D. Van Slyke, verbal communication.)

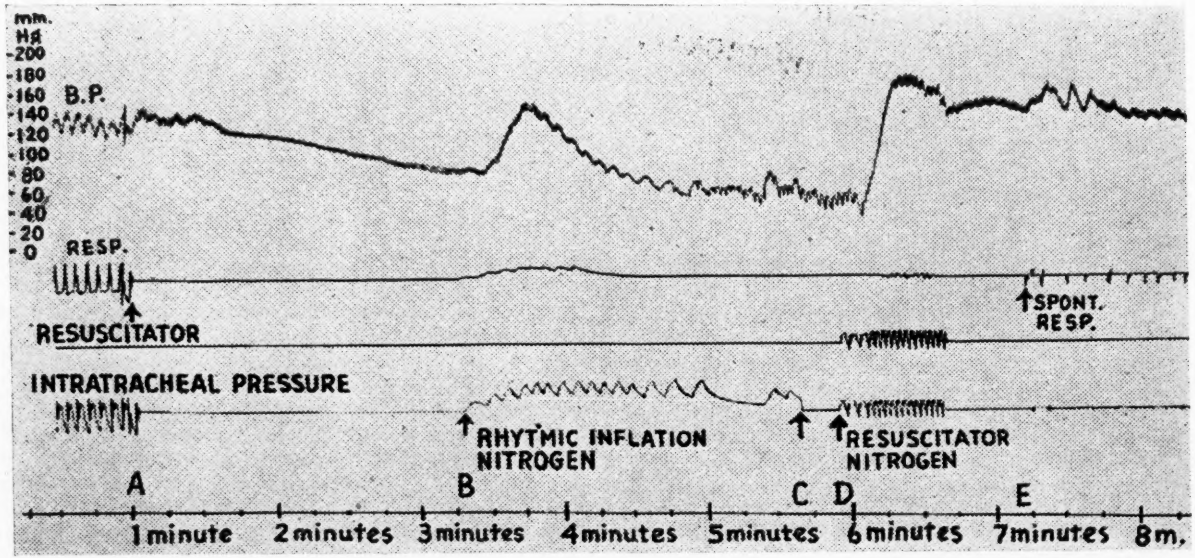

Fig. 11 (Dog 15).—Obstructive asphyxia at the face mask. *A*, Respiration ceases. At *B*, rhythmic inflation (plus 14 mm. Hg to 0) with nitrogen is started and is followed by a short-sustained rise of blood pressure and then a failure of the circulation to be maintained. The curve of the intratracheal pressure finds it difficult to reach the base line promptly when pressure is released, giving, therefore, the effect of insufflation rather than rhythmic inflation. This may indicate lack of tonus, spring, or elastic recoil of the chest wall in asphyxia. At *D*, the resuscitator with nitrogen is started and resuscitation follows. (Courtesy of J. A. M. A.)

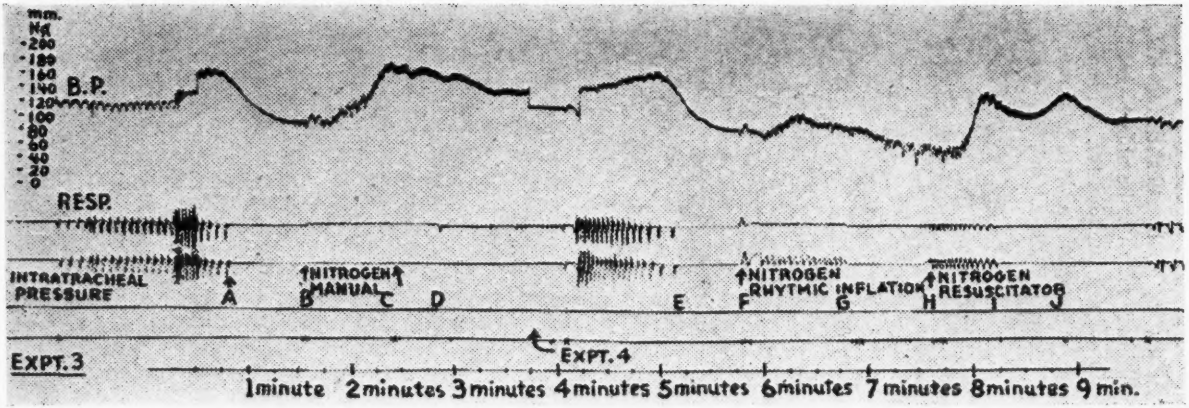

Fig. 12 (Dog 18).—Asphyxia by obstruction of intratracheal tube.

First half of graph (Experiment 3): *A*, Respiration ceases; *B*, manual artificial respiration with nitrogen inhalation maintained to point *C* is successful; *D*, spontaneous respiration starts.

Second half of graph (Experiment 4): After an interval of fifteen minutes, asphyxia was again carried out. *E*, Respiration ceases; *F*, rhythmic inflation of the lungs (plus 14 mm. Hg to 0) with nitrogen gives a small rise of blood pressure which is not maintained; *G* to *H*, the lungs are left connected to an atmosphere of nitrogen; *H*, the circulation is failing and the resuscitator with nitrogen is started and continued to point *I*; *I* to *J*, the lungs are still connected to an atmosphere of nitrogen. At *J* spontaneous respiration starts and animal is allowed to breathe air. (Courtesy of J. A. M. A.)

anoxia of the liver could lead to an accumulation of acid metabolites and liberation of acid proteins. The second is the role of the kidney in conserving alkaline base (Na, K) by substituting NH_3 in the salts it excretes. The anoxic kidney may not be able to hold back base or may allow the accumulation of acid metabolites. In asphyxia, deficient glucose oxidation with accumulation of beta-oxybutyric and aceto-acetic acids in the tissues and blood is to be considered also.

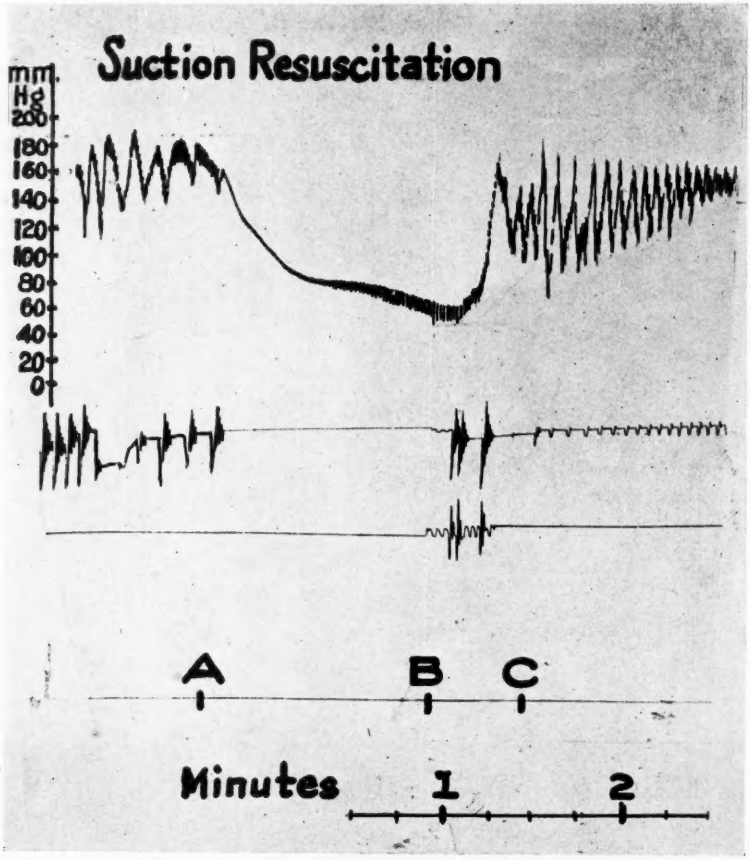

Fig. 13 (Dog 46).—Asphyxia by obstruction of the intratracheal tube. From above down, blood pressure, respiration, intratracheal pressure. *A*, Respiration has ceased; *B*, rhythmic suction on the lung (minus 8 mm. Hg pressure), alternating with oxygen inhalation (zero pressure), is started. Notice that the respiration is stimulated almost immediately and the blood pressure rises promptly. *C*, regular spontaneous respirations begin and the animal is allowed to breathe air. (Courtesy of Surgery.)

One of the important possible metabolites in asphyxia is lactic acid derived from anaerobic metabolism of glycogen in muscle and liver. This organic acid could not only help explain the fall of the total CO_2

of the blood; it could also explain the source of energy for the heart and brain centers during asphyxial (anoxic) resuscitation. This can be represented as follows:[1]

ENERGY FROM CONVERSION OF GLYCOGEN TO LACTIC ACID IN THE ABSENCE OF OXYGEN

Glycogen in muscle present up to 1 per cent
Muscle *contracting* in an atmosphere of nitrogen shows a quantitative decrease in glycogen and increase in lactic acid; when lactic acid reaches 0.3 per cent, muscle fails to respond.
Muscle *resting* in an atmosphere of nitrogen shows similar changes

$$(C_6H_{10}O_5)n \xrightarrow{\text{anaerobic metabolism}} (C_3H_6O_3)$$

glycogen → lactic acid plus energy

$$\text{lactic acid} + BHCO_3 = \text{Blactate} + H_2CO_3.$$

It was mentioned previously that the CO_2 content of the blood rises in asphyxia during the period of dyspnea and part of the period of cessation of respiration. It then declines. We offer the following possible causes to be considered in the rise and later fall of the total CO_2 content of the blood.

*Possible Causes of Rise of CO_2 Content of Blood.**—

1. Dyspnea causes increased work, metabolism, and a rise of CO_2 in the blood.

2. The continued rise of CO_2 in the blood after respiration has ceased is due to a lag, that is, the increased CO_2 in the tissues requires time to enter the blood stream, and the CO_2 in the blood stream cannot now be washed out by pulmonary ventilation.

3. In asphyxia there is more reduced hemoglobin than normal and this reduced hemoglobin can hold more CO_2 than oxyhemoglobin.

4. The increased stream into the plasma of gaseous CO_2 produced by increased metabolism (see Cause 1 in this list) and acid metabolites displacing CO_2 from bicarbonates (see Cause 4 in following list) is converted into alkaline bicarbonates (see preceding, Hamburger's phenomenon).

Possible Causes of Subsequent Fall of CO_2 Content of Blood.—

1. There is diminishing metabolism and fall of CO_2 in the tissues (from anoxia, shock, anesthesia, and loss of heat).

2. CO_2 is being gradually washed out from the circulating blood into the lung alveoli.

3. There is retention of CO_2 in tissues and capillary pools due to sluggish circulation and/or shock.

4. There is nongaseous acidosis (acid displaces CO_2 from bicarbonates):

   a. lactic acid from anaerobic metabolism of glycogen of liver and muscle

*According to Meyerhof,[12] muscle contracting under anaerobic conditions becomes at first *more alkaline*, due to the formation of the alkaline creatine. This may partly account for the initial high carbon dioxide content of the blood.

b. beta-oxybutyric acid and acetoacetic acid from diminished or absent glucose oxidation
c. acid proteins and/or other acid metabolites from deglycogenation and/or anoxia of the liver
d. retention of acid radicles and loss of base by anoxic kidney

5. CO_2 is being washed out of the blood by the resuscitator during asphyxial (anoxic) resuscitation

SUMMARY

It is seen that in both obstructive and nitrogen inhalation asphyxia there is first a rise, then a fall of total blood carbon dioxide content. Therefore, early in asphyxia and for a short time after the respiration has ceased, oxygen without carbon dioxide would appear indicated, theoretically, at least. Our interpretation of the fall of carbon dioxide content is that this fall represents, in part, an acidosis with an associated acarbia, or carbon dioxide deprivation. The acidosis itself might be stimulating to the cerebral centers and carotid body when present in conjunction with the administration of oxygen only. Whether the administration of carbon dioxide in addition to oxygen is actually detrimental would be difficult to say. There are two possible objections to the use of carbon dioxide in severe asphyxia: (1) Carbon dioxide in the presence of asphyxia is a respiratory (cerebral) depressant;[6, 15] (2) carbon dioxide is a peripheral vasodilator and can cause a fall of blood pressure in shock; in marked anoxia, shock is present.[6, 11]

However, in clinical application there is usually no way of knowing exactly the stage of asphyxia. The use of 5 to 7 per cent carbon dioxide with oxygen would, therefore, seem advisable to prevent and treat acarbia and to stimulate the cerebral centers.

We do not advocate rhythmic suck-and-blow resuscitation in asphyxia when respiration is present or after it has returned, for in these instances simple inhalation of oxygen, or carbogen, is usually indicated and sufficient.

Manual artificial respiration is a widely employed method and has its field of usefulness. However, it may be difficult or impossible in operations on the chest or in trauma to the abdomen, back, or chest. Where *both* lungs are collapsed from operative (accidental) or traumatic, open pneumothorax, rhythmic inflation with oxygen, or carbogen, would be indicated. Mouth-to-mouth inflation may also be a valuable emergency procedure. Aside from instances such as these, rhythmic inflation and suction with safe pressure has more to recommend it than other methods. Indeed, its use may save life where other methods would fail.[2-6]

CONCLUSIONS

1. Experimental evidence indicates that rhythmic inflation and suction of the lungs in advanced asphyxia, using safe pressures, is su-

perior to manual artificial respiration, rhythmic inflation alone, or rhythmic suction alone.

2. Rhythmic inflation and suction of the lungs has marked resuscitative power even when an inert (asphyxiating) gas is used, indicating the inherent superiority of the method.

3. Rhythmic inflation and suction is a more rapid and more effective stimulator of the circulation and respiration than the other methods tested.

4. Preliminary experimental evidence supports the hypothesis of acidosis and shock in advanced asphyxia, with a relative acarbia as an accompanying state.

5. The hypothesis is presented that in asphyxia, the anaerobic conversion of glycogen to lactic acid may supply limited energy for the heart and brain. This, together with the marked reflex stimulation it affords, could explain the effectiveness of asphyxial (anoxic) resuscitation in advanced asphyxia.

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ASPHYXIAL RESUSCITATION; THE PHENOMENON AND ITS MECHANISM

SAMUEL A. THOMPSON, M.D., F.A.C.S., AND GEORGE L. BIRNBAUM, M.D.*
NEW YORK, N. Y.

ASPHYXIA and resuscitation are of considerable importance in the operating room in civilian life, in industrial surgery, and of even greater importance in warfare, because of the tremendous increase in the casualties due directly or indirectly to asphyxia.

The importance of resuscitation has been recognized and a great deal of controversy still exists as to the value of various resuscitative methods. In an attempt to clarify this problem by experimental investigation, we observed a phenomenon which, to our knowledge, had not been previously described. This phenomenon has a very definite bearing upon resuscitation, and we have called it "the phenomenon of asphyxial resuscitation."[1-3] Briefly, it consists of the ability to restore the circulation and respiration in advanced asphyxia *without benefit of oxygen* through the use of rhythmic inflation of the lungs alternating with rhythmic suction deflation of the lungs, using an inert or asphyxiating gas. Paradoxical as it may seem, it is possible to resuscitate an animal in the third stage of asphyxia (cessation of respiration and falling blood pressure) by rhythmic inflation and deflation of the lungs, using the same inert (nitrogen, helium) gas which had just previously been used to produce the asphyxia (Fig. 1). Resuscitation with an inert gas by the same method is also possible in asphyxia which has been produced by obstruction (Fig. 2).

Asphyxia has been divided into four stages: initial apnea, dyspnea, cessation of respiration or terminal apnea, and arrest of the heart. Although it is convenient to consider that the total duration of acute asphyxia is approximately four minutes, with each stage occupying about a minute, the duration of each stage and the total time may vary. The stages can be summarized as follows:

Stage 1. Initial Apnea. The breath is held and the blood pressure rises.

Stage 2. Dyspnea. Breathing is labored. The blood pressure continues to rise. At the end of this period consciousness disappears. Tonic and clonic convulsions occur.

Stage 3. Terminal Apnea. The respiration ceases. Reflexes and muscle tonus disappear. The animal is relaxed and limp. The blood

*Major, Medical Corps, U. S. Army.
From the Laboratories of Surgical Research and the Thoracic Surgical Service of the New York Medical College, Flower-Fifth Avenue Hospitals.
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pressure falls progressively; toward the end of the stage the character of the pulse changes, the heart beat is irregular, and the blood pressure rapidly approaches zero. The sphincters are relaxed and the corneal and glottic reflexes are absent.

Stage 4. Arrest of the Heart.

It should be kept in mind that due to the fact that anesthesia was given and asphyxia produced in some cases by inhalation of inert gases, the stage of initial apnea may not be very evident, or even may be absent.

In evaluating resuscitation methods, it is necessary to consider the possibility of spontaneous resuscitation and at what point it may occur in asphyxia. It has been demonstrated by Coryllos[4] that if asphyxia of submersion is interrupted before the middle of the third stage, spontaneous resuscitation is always possible; he considered that in obstructive asphyxia "removal of the obstructing agent before the end of the third phase is followed as a rule by immediate spontaneous resuscitation. . . ." Since he considered his stages to occupy about a minute, this would place the critical point for spontaneous recovery under thirty seconds from the time respiration ceased in the case of submersion asphyxia and under forty-five seconds from the cessation of respiration in the case of obstruction asphyxia. Lougheed, Janes, and Hall,[5] in a study of asphyxia by drowning and tracheal obstruction, have found that after cessation of respiration there is a critical point at which the heart beat ceases to be expulsive. In general, the period between the point at which respiration ceased and the point at which the asphyxial procedure was stopped could not exceed eighteen seconds if spontaneous recovery were to take place. If this period was extended as little as a very few seconds longer, resuscitation even with manual artificial respiration, together with inhalation of oxygen carbon dioxide, was impossible.

We have shown that asphyxial resuscitation, using an inert gas, is possible far beyond the point in asphyxia where spontaneous recovery could take place (Fig. 1). Generally, we have started asphyxial resuscitation well beyond the forty-five second point following respiratory cessation, at a variable "critical point," where the heart action becomes altered and irregular (Fig. 2). The length of time which may elapse between respiratory cessation and the successful application of asphyxial resuscitation varies in each experiment; the longest interval was two and three-quarters minutes. In a classical experiment, the circulation recovers first, then respiration. The resuscitator* is usually kept in action until spontaneous respiration has taken place, although this is not always necessary. Once the resuscitator has produced recovery of the circulation, as indicated by a marked and sus-

*Emerson Resuscitator.

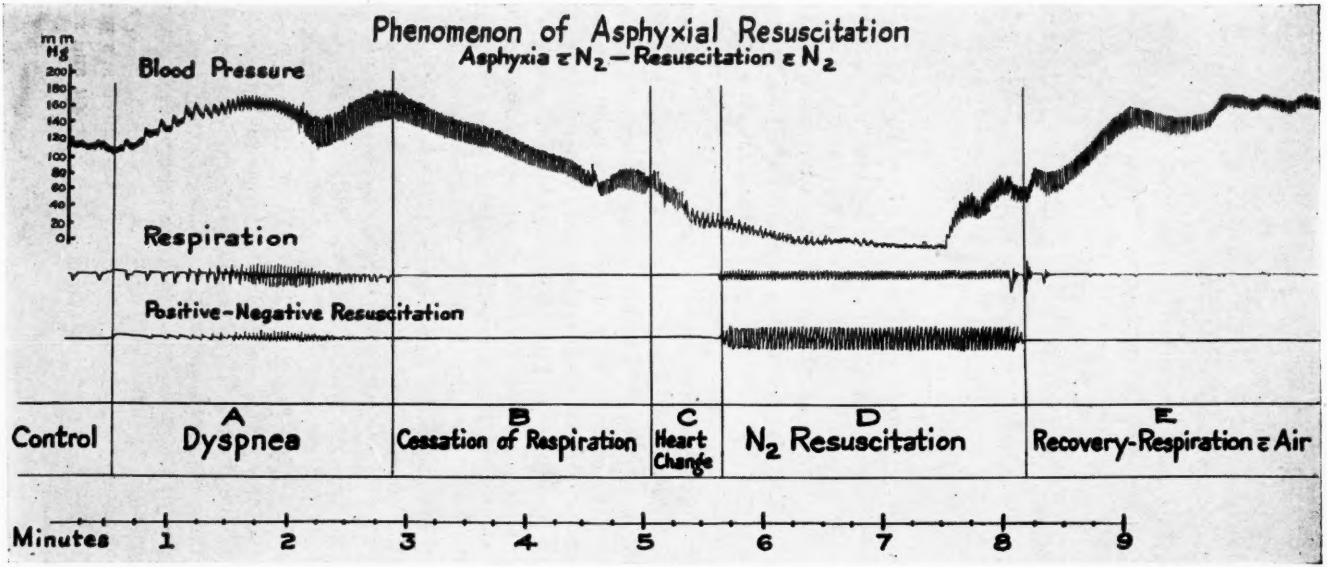


Fig. 1.—The phenomenon of asphyxial resuscitation. Nitrogen inhalation asphyxia is produced by way of a leakproof intratracheal tube. After cessation of respiration and in the period of cardiovascular changes with a rapidly falling blood pressure, the lungs are rhythmically inflated and deflated with the same nitrogen which had just produced the asphyxia by means of a mechanical resuscitator of the suck-and-blow type.

A. Inhalation of 100 per cent nitrogen. This is the period of dyspnea. *B*, At the beginning of this period respirations have ceased. The blood pressure begins to fall. *C*, The heart beat is slower and somewhat irregular and the blood pressure is falling rapidly. *D*, Nitrogen resuscitation is started as described above, two and three-quarter minutes after respiratory cessation. The blood pressure continues to fall and approaches zero, approximately one and three-quarter minutes after nitrogen resuscitation was started. There is resuscitation of the cardiovascular system, as shown by the sudden and continued rise of the blood pressure. Spontaneous respirations occurred later and the resuscitator was discontinued. The intratracheal tube at the end of *D* is exposed to the atmospheric air for the first time since nitrogen inhalation was started, approximately eight and one-half minutes. *E*, Recovery. (Courtesy of J. A. M. A.)

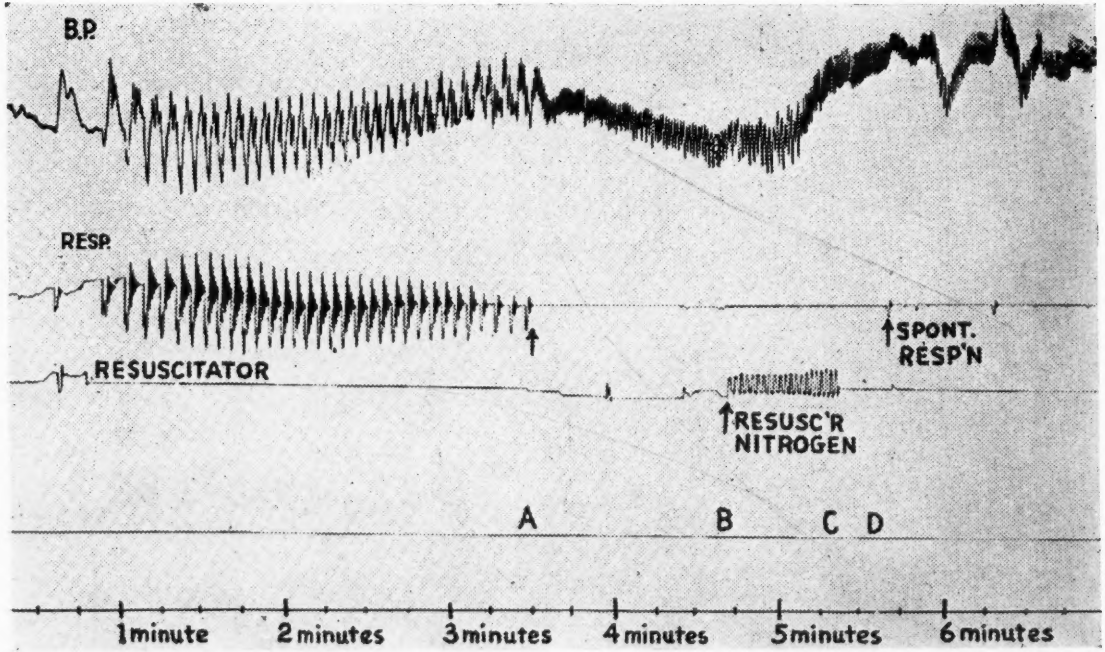


Fig. 2.—Asphyxial resuscitation (nitrogen) following obstruction asphyxia. Nitrogen resuscitation with a mechanical resuscitator was started approximately one and one-quarter minutes after respiratory cessation. The resuscitator was discontinued after cardiovascular resuscitation was established, but before spontaneous respirations had occurred.

A, Obstructive asphyxia is accomplished by clamping the intratracheal tube, and respirations ceased at *A*. *B*, Nitrogen resuscitation started. *C*, Cardiovascular resuscitation is apparent and the resuscitator is discontinued. *D*, Spontaneous respirations occur and the intratracheal tube is opened to atmospheric air. (Courtesy of J. A. M. A.)

tained rise of the blood pressure, spontaneous respirations can occur even when the resuscitator has been discontinued (Fig. 2).

We now propose to present the technique of the experiments, the experimental data, and a detailed analysis of this phenomenon.

TECHNIQUE

Dogs weighing 8 to 12 kg. were anesthetized by the intraperitoneal injection of veterinary nembutal, 1 c.c. per 5 pounds. The animal was then placed on its back. In the first few experiments, a pharyngeal airway was inserted and a specially fitted metal face mask with inflation cuff on its rim was applied. In the remaining experiments, an intratracheal tube surrounded by an inflatable rubber cuff was inserted to make the trachea leakproof. Asphyxia was produced by either completely obstructing the face mask or clamping the intratracheal tube. In other experiments, asphyxia was produced by inhalation of inert gases such as nitrogen or helium.

Tracings on the kymograph were produced by recording mercury manometers which registered the blood pressure, respiration (intrapleural pressure), intratracheal pressure, and resuscitator pressures. Blood pressure was recorded from the cannulized femoral artery. Intrapleural pressure or respiration was recorded by means of a fine brass intrapleural cannula.

For mechanical resuscitation the Emerson resuscitator was used. This apparatus inflates to 14 mm. Hg pressure and aspirates to minus 9 mm. Hg pressure; it was usually regulated to make sixteen to twenty complete excursions or respiratory strokes per minute. It was connected when needed to the face mask or intratracheal tube, as the case might be. Where it was desired to use rhythmic pressure alone or rhythmic suction alone on the lungs, the desired pressures were regulated by underwater glass tubing. Rhythmic pressure with a gas was given at plus 12 to 14 mm. Hg pressure with release to 0 mm. Hg pressure (atmospheric pressure) of the gas. Rhythmic suction on the lung was carried out at minus 8 to 12 mm. Hg pressure with release to 0 mm. Hg pressure (atmospheric pressure) of the gas. Manual artificial respiration was done by bilateral manual compression of the chest by the four fingers and palms of the hands, at the same time that the thumbs and thenar eminences compressed the abdomen over the epigastric region; it was carried out moderately vigorously, the lungs being in communication with an atmosphere of the particular gas in question.

Inhalation asphyxia by inert gas was carried out with an arrangement to prevent rebreathing of carbon dioxide, since this latter gas would make the problem more complex. We called this "blow-off" inhalation asphyxia, and it was accomplished by leading the expired air through the expiratory tube of the inhalator under 1 cm. depth of water.

Vagus section was performed in the midneck. Carotid sinus denervation was done by exposing the bifurcation of the common carotid artery below the angle of the jaw and stripping 1 cm. of the adventitia away from the fork of the common carotid, from each of the following: common carotid, external carotid, and internal carotid artery. The angle of the fork itself was stripped and denervated. The stripped areas were painted with two applications of a 5 per cent alcoholic solution of phenol and then swabbed clean with alcohol.

EXPERIMENTAL DATA

The experimental data are tabulated in Table I for ready reference.

TABLE I

EXPERIMENT	FIG. NO.
Central depressing effect of carbon dioxide in asphyxia	3
Failure of asphyxial resuscitation after bilateral vagotomy	4
Successful, but altered and sluggish asphyxial resuscitation in obstructive asphyxia following bilateral carotid sinus denervation	5
Failure of asphyxial resuscitation in nitrogen inhalation asphyxia following bilateral carotid sinus denervation	6

Before analyzing the possible factors involved in resuscitation without benefit of oxygen, it may be well to recall a few important points in the physiologic control of circulation and respiration.

It is known that afferent impulses passing by way of the carotid sinus nerves, the aortic and vagus nerves to the medulla exert an influence upon the vasomotor center, the cardiac center, the respiratory center, and upon adrenaline secretion. In fact, electric action currents can be recorded in these nerves.

The carotid sinus nerves, the aortic and vagus nerves form a closely functioning group which, in general, exerts a reflex influence on the vasomotor and respiratory centers. Because of this regulation, the sinus and aortic nerves together are referred to as buffer nerves by Wright;[6] they are sensitive to changes of pressure as well as to chemical changes.

The most generally known reflex by way of the vagus is that of Hering and Breuer.[7] In the experiments of Head, referred to by Wright, forceful inflation of the lung causes a reflex expiratory effort, a forceful deflation, or suction, on the lung (and not a passive expiration), causes a reflex inspiratory effort. These experiments only partly confirm the Hering-Breuer reflex which presupposes both inspiratory and expiratory fibers in the vagus; at the height of inspiration the latter is stopped by inhibitory impulses from the lung to the respiratory center; at the normal depth of expiration the latter is stopped by impulses to the respiratory center which initiate inspiration. However,

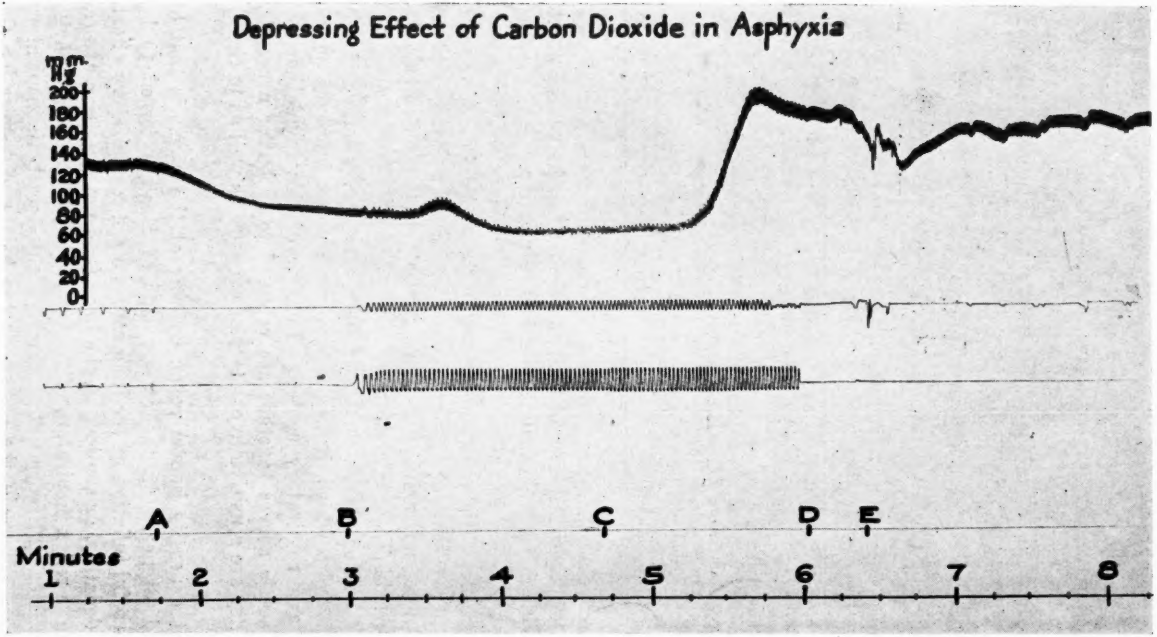


Fig. 3.—Depressing effect of carbon dioxide in asphyxia. An inert gas (100 per cent nitrogen) used with positive-negative resuscitator is successful after failure with 90 per cent nitrogen and 10 per cent carbon dioxide. Carbon dioxide, in the presence of asphyxia, depresses the cerebral centers and apparently prevents resuscitation by an inert gas. Through a leak-proof intracheal tube, nitrogen inhalation asphyxia has been given with an arrangement for blow-off of the expired air in order to prevent carbon dioxide accumulation. At *A*, the respiration has ceased; *B*, resuscitator started using nitrogen with 10 per cent carbon dioxide; *C*, pure nitrogen is used with the resuscitator; *D*, resuscitator off, tracheal tube is connected with nitrogen; *E*, spontaneous respiration starts (resuscitator pressure: plus 14 mm., minus 9 mm. Hg).

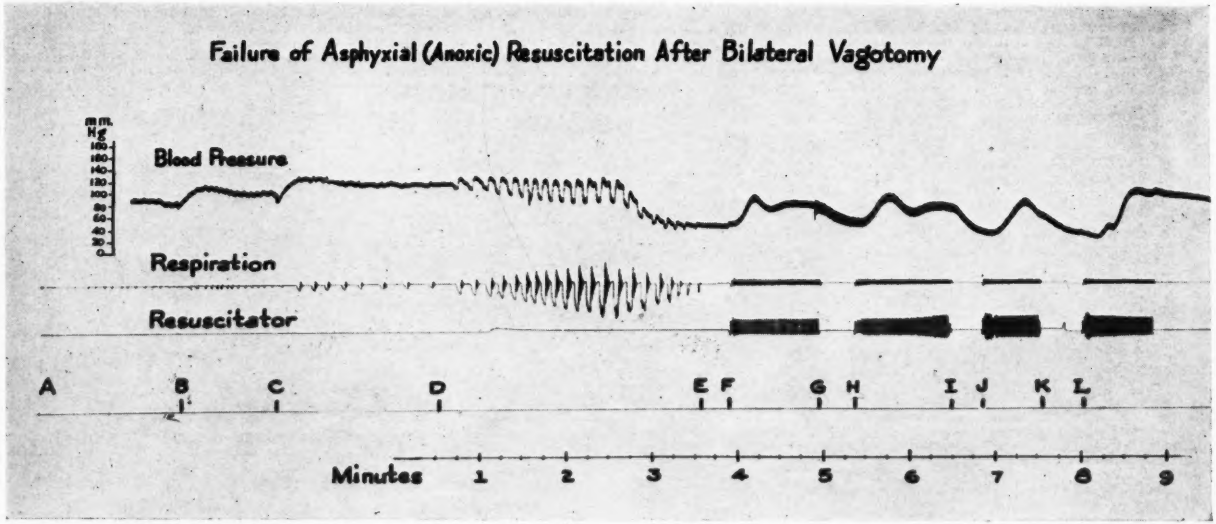
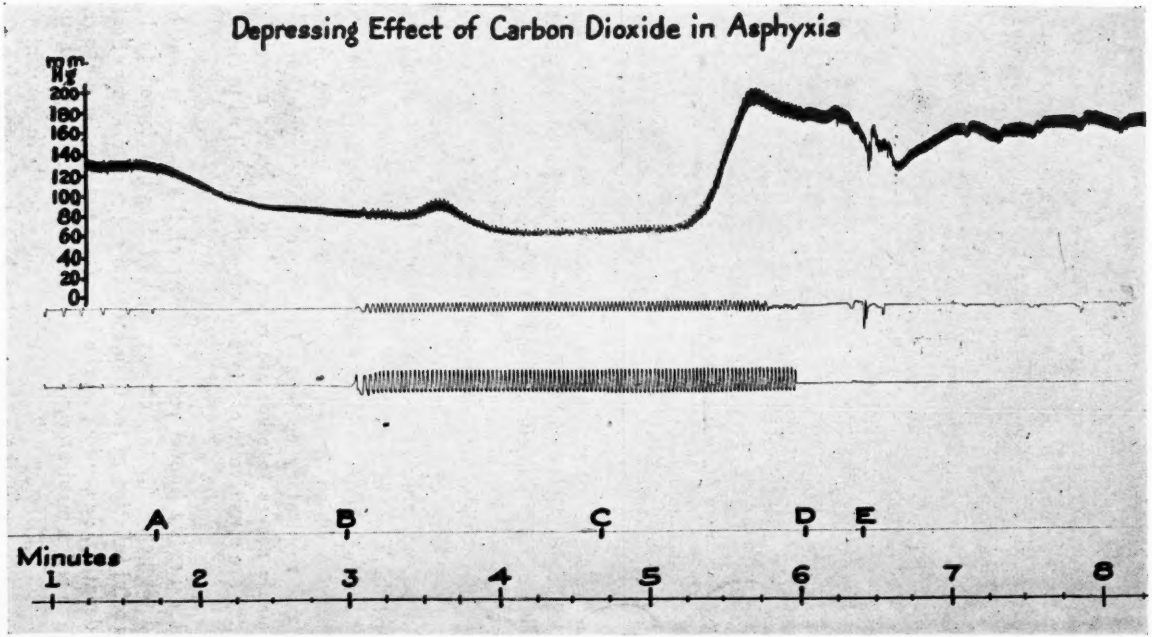


Fig. 4.—Failure of asphyxial (anoxic) resuscitation after bilateral vagotomy. *A*, Control before vagotomy; *B*, left vagus is cut in the midneck; *C*, right vagus cut in the midneck; *D*, asphyxia begun by clamping intratracheal tube. *E*, respiration ceases; *F* to *G*, resuscitator with nitrogen applied; *G* to *H*, tracheal tube connected to nitrogen only; *H* to *I* and *J* to *K*, resuscitator with nitrogen reinstituted, but the blood pressure could not be maintained and spontaneous respirations did not occur. Following this the animal was resuscitated with oxygen, beginning at *L*. (Courtesy of Surg., Gynec. & Obst.)



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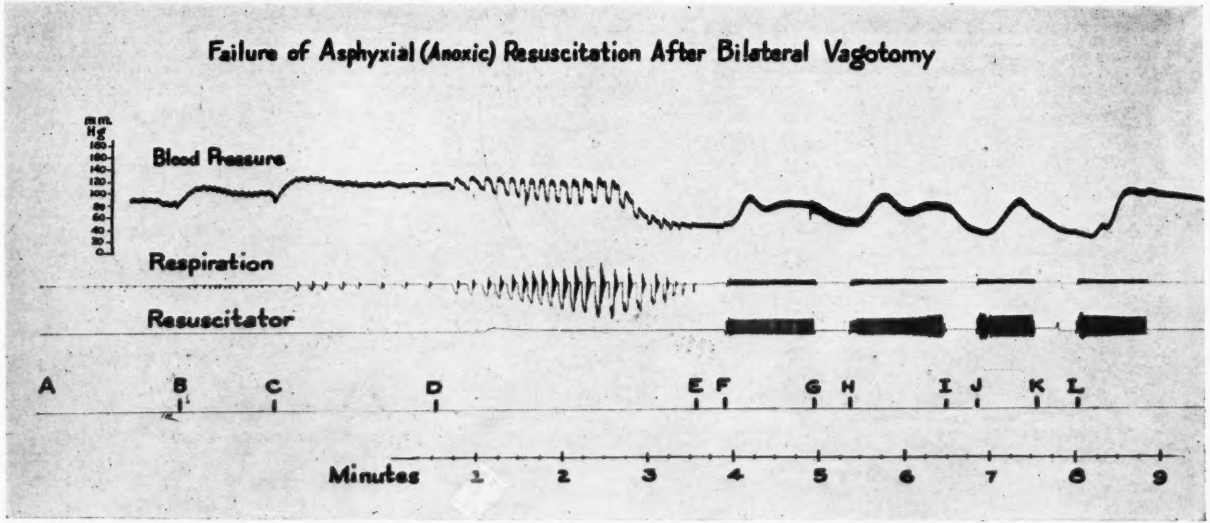

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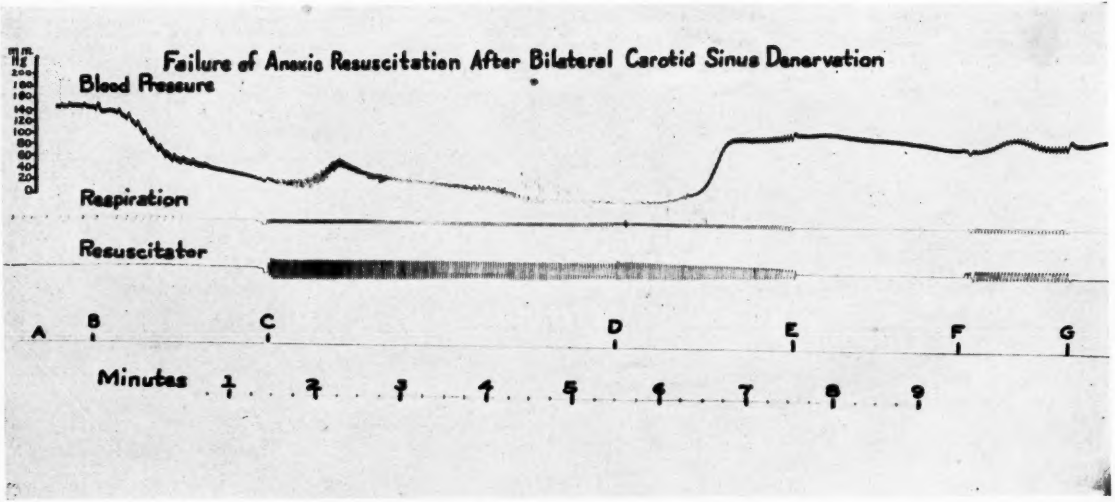

Fig. 5.—Failure of asphyxial resuscitation after bilateral carotid sinus denervation. *A*, After denervation. *B*, Through a leakproof intratracheal tube, nitrogen inhalation asphyxia has been started with an arrangement for blow-off of the expired air in order to prevent carbon dioxide accumulation. Notice the sharp fall of the blood pressure even before the respiration ceases. *C* to *D*, Resuscitator with nitrogen fails to resuscitate. *D* to *E*, Resuscitator with oxygen restores the circulation. *E* to *F*, Trachea connected with oxygen. *F* to *G*, Resuscitator with oxygen started again in an attempt to initiate spontaneous respiration. (Courtesy of Surg., Gynec. & Obst.)

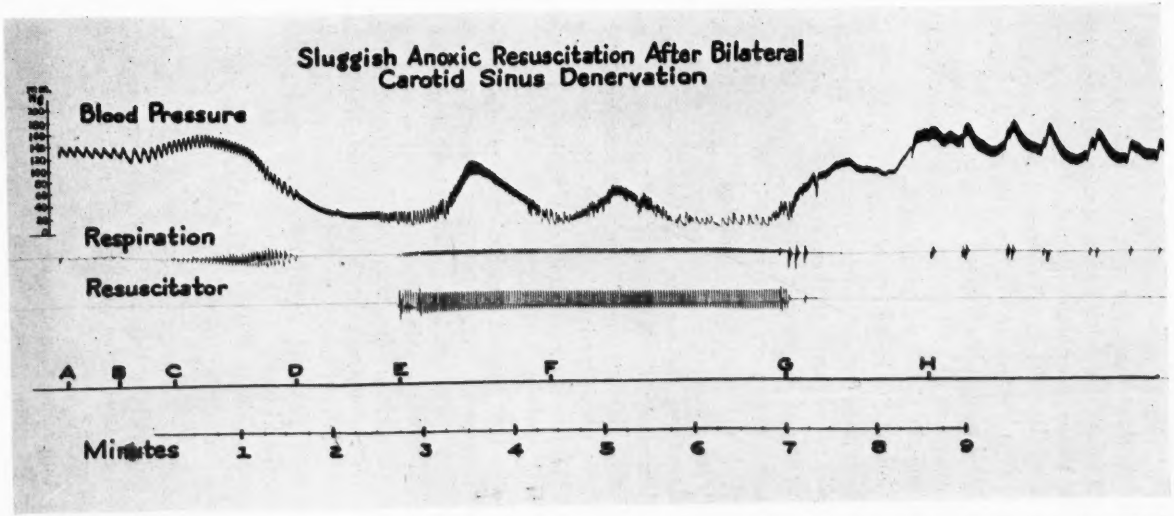

Fig. 6.—Sluggish asphxial resuscitation following bilateral caro'id sinus denervation. *A*, After carotid sinuses were denervated. *B*, Asphyxia started by clamping the intratracheal tube. *B* to *C*, Period of primary apnea. *D*, Respirations cease. *E*, Resuscitator with nitrogen started. *F*, The blood pressure falls and the resuscitator is continued. *G*, Spontaneous respirations occur, the blood pressure is elevated, the resuscitator is discontinued and the tracheal tube is opened to atmospheric air. (Courtesy of Surg., Gynec. & Obst.)

if the vagus is cut, action currents can be detected in the distal cut end of the nerve on inflation and suction and not on mere passive expiration.

According to Adrian[8] there are sense, or end, organs in the lung called stretch receptors which give rise to measurable action currents. They are unaffected by anesthetics or lack of oxygen, and circulatory failure has no immediate effect. Their function is to signal the volume of the lung at each movement and they continue to do this for one-half hour or more after the heart has stopped beating.

Normally, these end organs are active only on inspiration or inflation of the lungs and are the slowly adapting type.

Suction deflation of the lungs stimulates a new set of end organs which are not stimulated by the normal respiratory movements of the lungs and there is no evidence of their activity in moderate dyspnea. These end organs are of the rapidly adapting type and, from our experimental evidence, are more powerful and effective in resuscitation than are the stretch receptors of inflation. Forceful deflation of the lungs by external compression of the thorax in some animals is not enough to stimulate these end organs, which react quite readily to suction. The impulses aroused by inflation of the lungs inhibit the inspiratory cells of the respiratory center and those aroused by deflation excite them. These considerations emphasize the importance of suction, as well as inflation, in resuscitation procedures to stimulate reflexly the medullary centers.

There is evidence that central irradiation from one medullary center to the other may occur. Thus, in the study of sinus arrhythmia, Heymans (Wright, p. 305) concluded that this phenomenon is due to direct irradiation from the respiratory to the cardiac center ("isolated head" experiment). Conversely, it would seem that irradiation from the cardiac and/or vasomotor centers to the respiratory center can occur, for it will be noticed in our experiments that, in asphyxial resuscitation with inert gas, the circulation recovers regularly before spontaneous respiration begins.

Several hypotheses presented themselves to us to explain asphyxial resuscitation; thus, it could be due to:

1. Massage action on heart and great vessels in the thorax, the intrapleural and mediastinal pressures varying synchronously with the resuscitator pressures; "milking" action on alveolar capillaries.*

2. Mobilization of unused oxygen sources which might still be present in the blood vessels and tissues, by initiating a return of the circulation as described under hypothesis 1.

*We believe that the factors mentioned in hypothesis 1 have a very definite beneficial effect on the circulation.

3. A washing out of carbon dioxide from the blood by the resuscitator, thus removing a substance depressing to the medullary centers in the presence of asphyxia.

4. A reflex mechanism to the medullary centers, by way of the vagus or other nerves, concerned with circulation and respiration.

5. A combination of two or more of these.

We have seen that with nitrogen resuscitation the circulation recovers regularly before spontaneous respiration begins. Three hypotheses present themselves:

1. The afferent reflex impulses go first to the cardiac and/or vasomotor centers and irradiate to the respiratory center which is at first refractory.

2. The nerve impulses first reach the respiratory center, which as yet does not respond, and irradiate to the cardiovasomotor centers.

3. The impulses reach the centers simultaneously, the respiratory center being refractory at the start.

Once the circulation is more or less definitely re-established by suck-and-blow resuscitation with inert gas, the animal may be kept alive for one, two, three, or more minutes, as the case may be, if the procedure is persisted in instead of discontinuing the resuscitator. This is quite remarkable, since, when the respiration ceases in asphyxia, the oxygen saturation of the blood is already at an extremely low level.

Blood gas analyses also show a high oxygen unsaturation (low oxygen content) at the institution of asphyxial resuscitation; at later periods of the procedure, the venous oxygen unsaturation increases slightly and this could indicate utilization of oxygen by such vital organs as the heart and brain, or that some of the oxygen in the pulmonary capillaries is being washed out into the alveoli and aspirated into the resuscitator. The oxygen in the larger blood vessels is the first available; but mobilization of oxygen from capillary pools and the tissues is a possibility to consider. The washing out of carbon dioxide from the blood into the alveoli and resuscitator, by lessening medullary depression, may also be a factor in sustaining and prolonging life.

Bilateral vagus section in the midneck prevents resuscitation with inert gas (Fig. 4). Carotid sinus denervation may (Fig. 5) or may not (Fig. 6) prevent resuscitation. It is, therefore, evident that the phenomenon is primarily a reflex mechanism (hypothesis 4) in which the vagus (cardio-aortic, pulmonary fibers, or both) is indispensable. The carotid sinus nerve is concerned but not always indispensable and its role may, therefore, be that of a regulator sending impulses to the medullary centers.

It thus appears that the mechanical inflation and deflation of the lungs by means of a suck-and-blow resuscitator with an inert gas brings into play two powerful reflex stimulants to the cardiovascular and respiratory centers. Of these reflexes the one occurring on suction deflation of the lungs appears to be the more powerful. This reflex does not appear to be stimulated by passive expiration as obtained by inflation and release of the lungs. It does not appear to be stimulated by manual artificial respiration, but it is quite regularly stimulated by suction deflation of the lungs.

This suction phase of resuscitation has not only not been emphasized by most authors, but has been de-emphasized as being unnecessary or dangerous, or unlike natural respiration. It should be emphasized here that this powerful reflex is not, and cannot be, stimulated by normal respirations.

Asphyxial resuscitation is certainly not recommended for clinical application, since the use of oxygen gives far superior results.

The practical application of the phenomenon of asphyxial resuscitation reveals the importance of using suction as well as inflation of the lungs in respiratory resuscitation.

SUMMARY

In our experiments we have produced in dogs various stages of asphyxia by obstruction of the trachea, or by inhalation of inert (asphyxiating) gases such as nitrogen or helium. If the asphyxiating procedure is not stopped within twenty to thirty seconds after the cessation of respiration, spontaneous recovery does not occur. However, if within periods of forty-five seconds to two and one-half minutes after cessation of respiration, suck-and-blow resuscitation with inert (asphyxiating) gas is applied, resuscitation of the circulation and respiration can be accomplished in 85 per cent of instances. The addition of 10 per cent carbon dioxide to the inert gas inhibits resuscitation by depression of the medullary centers. Bilateral vagus section before asphyxia is started prevents resuscitation with inert gas. The carotid sinuses are also concerned in this phenomenon. Asphyxial resuscitation is primarily a reflex phenomenon which is initiated most efficiently by suck-and-blow resuscitation.

CONCLUSIONS

1. In asphyxia with respiratory cessation, a powerful reflex mechanism is stimulated by the mechanical inflation and suction deflation of the lungs. This reflex is important and has a direct influence upon resuscitation.

2. The mechanism of this phenomenon has been analyzed.

3. Clinically, in advanced asphyxia where the respiration has ceased, advantage should be taken of the phenomenon by the administration

of oxygen or oxygen carbon dioxide by the suck-and-blow type of resuscitation within safe limits of pressure.

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DIVERTICULUM OF THE RIGHT PRIMARY BRONCHUS

JOSÉ ARCE,* M.D.
BUENOS AIRES, ARGENTINA

A DIVERTICULUM of the bronchi is a rare finding. When the lesion does occur it is demonstrated best by bronchography with lipiodol. This is not a routine examination in the daily practice of thoracic surgery, but must be employed in some cases, especially when the diagnosis presents some difficulties.

CASE REPORT

Patient No. 94749 (Registry of the Institute of Surgery).—X-ray films revealed an irregular shadow in the upper lobe of the right lung (Fig. 1). Our first impression was that the lesion was a complicated hydatid cyst, because the patient related having coughed up "saline water" and "white flesh," a common manifestation when lung echinococcus are expelled by coughing. If the parasitic membranes are not expelled or only partly evacuated they remain incarcerated in the adventitial sac. This will cause the shadow on x-ray films to diminish in size and on the lateral projection the lesion assumes a polygonal shadow in contrast to the former spherical outline. The diagnosis is not, however, easy to establish and it is necessary to undertake other radiologic studies. Bronchography with lipiodol, when proper technical precautions are observed, will usually demonstrate the oil in the adventitial pouch, thus revealing the bronchial opening, and show a typical shadow around the parasitic membrane.

An examination with the bronchoscope was undertaken in our patient and after observing the normal carina we could see on the inside of the right main bronchus another complete abnormal sagittal spur. This "new carina" was situated in front of the right upper lobe bronchus. Believing that it could be an aberrant bronchus we introduced a catheter into the opening and injected lipiodol. The first film showed the bronchial tree and the x-ray shadow previously described, but no lipiodol entered the supposed adventitial pouch (Fig. 2).

However, in one of the x-rays taken later we could see a small shadow beside the inner wall of the right main bronchus (Fig. 3) in a position which corresponded to the abnormal spur found during the examination through the bronchoscope. The small shadow was bullet shaped. The x-ray also showed the original shadow without modification (Fig. 3).

*Professor of Thoracic Surgery, University of Buenos Aires.
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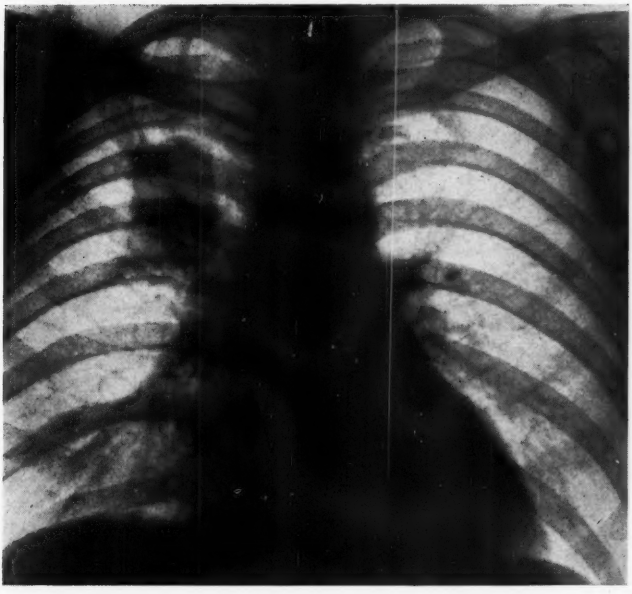

Fig. 1.—Routine x-ray showing shadow of incarcerated echinococcus membrane in the upper right lobe.

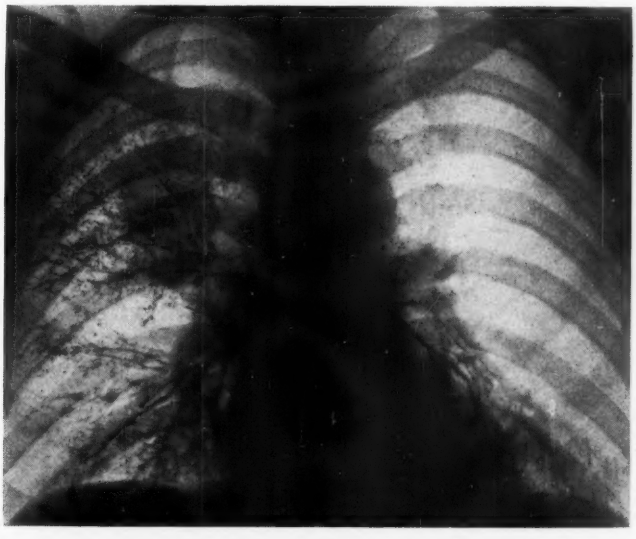

Fig. 2.—Bronchography showing same shadow.

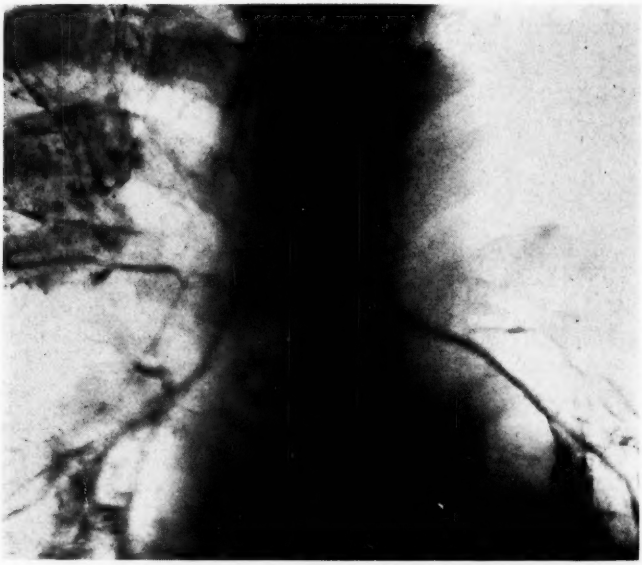

Fig. 3.—Special x-ray showing two shadows: the diffuse one of the incarcerated membrane and the bullet-shaped one corresponding to the bronchial diverticulum.

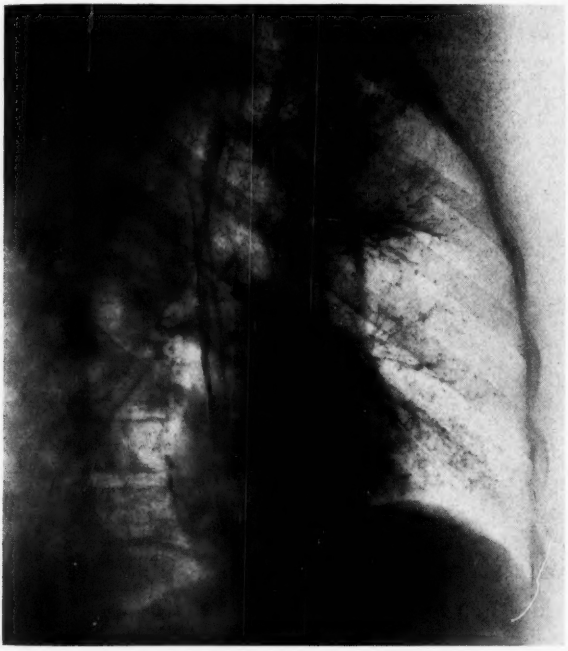

Fig. 4.—Lateral views showing the vertebra, esophagus, trachea and bronchi, and diverticulum.

The lateral projection (Fig. 4) revealed that the bullet-shaped shadow corresponded to the diverticulum of the right main bronchus. The horizontal level of the opacity (at the base of the shadow) points to its large communication with the bronchus. In the same x-rays the vertebral arch, esophagus, trachea and its bifurcation, and the shadow of the lesion for which the studies were undertaken can be seen clearly. The enlarged center of Fig. 5 shows a splendid outline of the diverticulum.

Fig. 5.—Enlarged center of former illustration, showing diverticulum.

At operation it was demonstrated that the large shadow corresponded to an incarcerated parasitic membrane. Four weeks after its removal the patient was able to leave the Institute completely recovered.

Even though the diverticulum of the right main bronchus was not of clinical importance it is advisable to bear the lesion in mind because a diverticulum may be the seat of a tumor just as any other part of the bronchial wall.

CORONARY LIGATION IN WOUNDS OF THE HEART

Report of a Case in Which Ligation of the Anterior Descending Branch of the Left Coronary Artery Was Followed by Complete Recovery

E. de Jesus Zerbini, M.D.*
Sao Paulo, Brazil

SINCE 1896, when Rehn first successfully operated upon a patient with a penetrating wound of the right ventricle, approximately 600 cases of wounds of the heart have been reported. The pathology of these dangerous wounds and their treatment have already been discussed, the best reports having come from American surgeons (Beck, Bigger, Elkin, and others).

Ligation of a branch of the coronary artery in the treatment of a myocardial wound is not an infrequent occurrence but the reported cases are rare. In this article a case of ligation of the anterior descending branch of the left coronary artery with complete return to normal is being reported. At the time of this communication, the patient shows no diminution of cardiac reserve nor heart pain; the electrocardiogram shows no signs of myocardial infarction, and there are not the abnormalities in lead IV which were observed by Olim and Hughes in a similar case reported in 1939.[5]

The branches of the left coronary vessels are more frequently injured than those of the right because of their more exposed position on the anterior surface of the heart. According to the standard books on anatomy, the arteries of the heart are considered as vessels which do not anastomose among themselves. Ligation of one of these arterial branches should, therefore, always be followed by anemic infarction of the area supplied by that vessel. The idea of terminal vessels was originated by Cohnheim in 1872, but, in discussing the absence of arterial anastomoses between two coronary vessels, he fails to consider the capillary anastomoses. There are liberal anastomoses throughout the capillary network of the body, even in the brain (Seep and Lochi), and in the kidneys and heart, as shown by Iwanow.[4] Ligation of an arterial branch may or may not be followed by infarction of the corresponding area, depending upon the anatomic and functional condition of the capillary network. In the heart, ligation of a coronary artery always is a cause for a grave prognosis, but infarction is not inevitable. Iwanow reported an interesting experimental study of the collateral circulation in the heart. He ligated the branches of the

*Assistant Professor of Surgery, Sao Paulo University School of Medicine, Sao Paulo.
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coronary arteries in dogs and later, after sacrificing the dogs, injected a radiopaque substance into the vessels and took roentgenograms. By means of these arteriograms he demonstrated that a coronary branch, after its double ligation at two different points, could become filled by way of the collateral circulation.

There have been cases of coronary ligation in which the typical electrocardiographic curves of infarction have been noted and at autopsy aneurysms of the corresponding areas have been found (Gronwald). The electrocardiographic tracings of an anterior infarction after the ligation of a descending branch of the left coronary artery was reported by Davenport, in 1926, and later by Elkin and Phillips,[3] and by others. There is another group of cases in which the typical electrocardiographic curves of infarction do not correspond to the clinical evidence of infarction (Olim and Hughes[5]). In a third group of patients, the myocardial wound has been sutured without injury to a coronary vessel and yet the same electrocardiographic tracings as seen in infarction have been noted (Mondry). Finally, there is a group in which ligation of an important coronary branch has been followed neither by clinical signs of infarction nor by electrocardiographic evidence, as in the case being reported and in others in the literature. After ligation of a coronary branch, each patient reacts according to the size of the vessel and to the condition of the collateral circulation.

In the patient whose case is being presented, it was observed that the injury involved the anterior descending branches of the left coronary artery and vein, and they were ligated. Fourteen days after operation, an electrocardiogram showed only a little flattening of the T wave which corroborated the clinical evidence of a pericarditis with effusion. In the second electrocardiogram, taken forty-six days later, T was inverted in leads II and III, and ST showed a slight upward convexity followed by an inverted T wave, which is the earliest electrocardiographic evidence of myocardial infarction (Pardee pattern). However, these changes in the tracings were less evident in the third record and were not seen in the fourth where T had regained its upright position in leads I, II, and III but was still inverted in IV. These electrocardiographic changes must have been related to the pericarditis (Fig. 3) as they disappeared after its treatment. The fifth record, taken 238 days after operation, is practically normal except for T_4 which remains inverted. Here, however, it can be seen that there are no typical curves of myocardial infarction. Furthermore, the changes in the fourth lead and the absence of Q_4 which are seen in experimentally produced anterior myocardial infarctions, as observed originally by Davenport and later reported by Olim and Hughes, are not present here. By coincidence, the site of ligation of the coronary vessel in the case of Olim and Hughes was exactly the same as in the case herein reported.

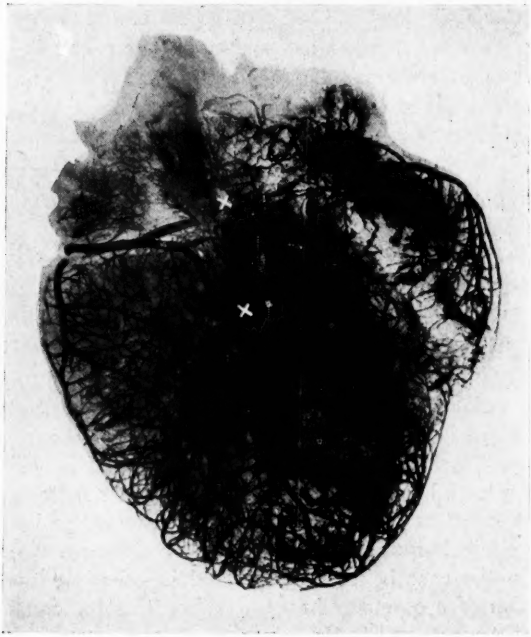

Fig. 1.—Coronary arteriography after ligation of a coronary branch in two different places. The segment of the vessel between the two ligatures is filled through the collateral circulation (Iwanow).

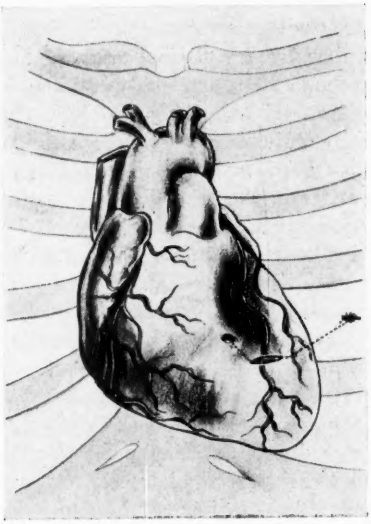

Fig. 2.—Sketch of the heart illustrating the site of the laceration. There is a perforation of the right ventricle and an injury to the left anterior descending coronary vessels.

CASE REPORT

D. Z., a 6-year-old boy, was seen Feb. 26, 1942, fifty minutes after having been injured by an iron splinter which had penetrated the fourth intercostal space to the left of the sternum. The patient was comatose with imperceptible pulse and blood pressure. The superficial veins, especially in the neck, were distended, and the venous pressure was found to be 20 cm. of water. He was cyanotic. The wound was in the fourth interspace, 2 cm. from the left border of the sternum. Heart sounds could be heard only with difficulty.

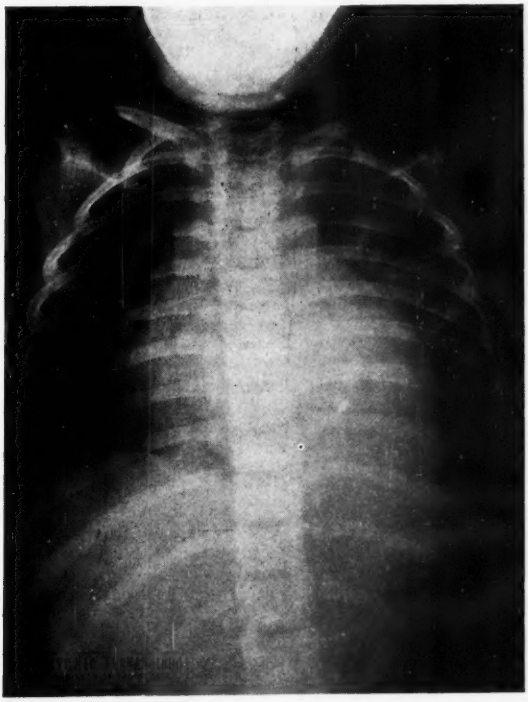

Fig. 3.—Postoperative roentgenogram showing the enlarged cardiac shadow which was present during the course of the pericarditis with effusion.

On fluoroscopic examination the iron fragment could be seen within the cardiac shadow, and signs of cardiac tamponade were present.

Operation was carried out through an incision in the bed of the fourth left rib after resection of its anterior arch. A perforation of the pleura was seen, and the lung was collapsed. There was an opening in the pericardium which was enlarged and blood and clots were removed, revealing a laceration 1.5 cm. long on the anterior surface of the right ventricle which reached the anterior descending branches of the left coronary vessels at a point about 4 cm. from the apex (Fig. 2).

The cardiac tamponade was relieved as soon as the blood and clots escaped from the pericardial sac. The laceration was closed with three interrupted sutures of silk, and the injured coronary branches were ligated. Immediately after the relief of the tamponade there was improvement in the pulse, and the blood pressure was elevated.

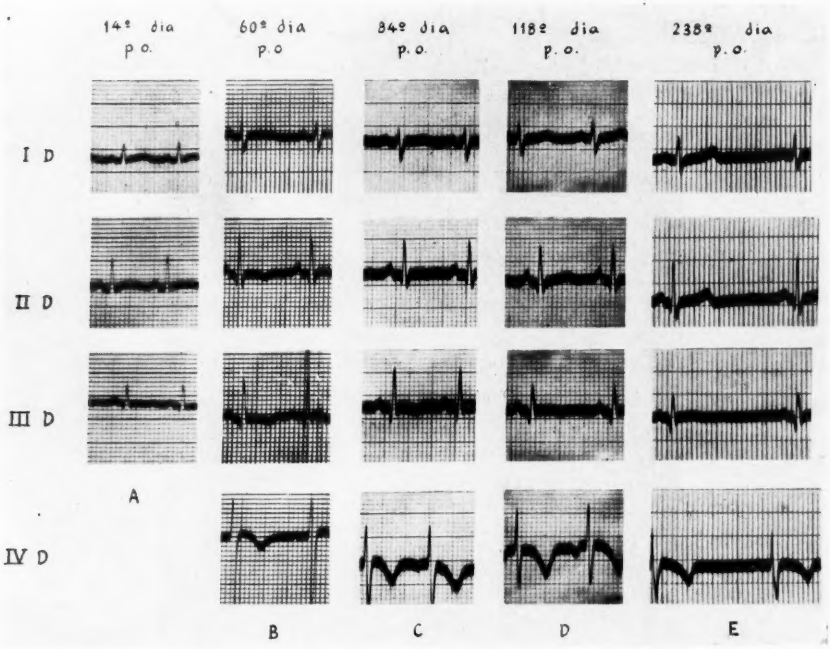

Fig. 4.—Serial electrocardiograms. Record *A* taken 14 days after operation with a little flattening of the T wave; record *B*, taken 60 days after operation, with an inverted T in Leads II and III; record *C*, 84 days after operation; record *D*, 118 days after operation; and record *E*, 238 days after operation, showing an inverted T4.

During the postoperative period the clinical picture of traumatic pericarditis was observed and the roentgenograms revealed the presence of a large amount of fluid in the pericardial sac (Fig. 3). At this time the electrocardiogram showed changes supporting this diagnosis (Fig. 4). The patient was kept at absolute rest in bed for two months and was treated by means of repeated aspirations of fluid from the pericardial cavity (Fig. 5). After two months he was discharged feeling perfectly well. Since then he has been examined at weekly intervals and one year after the operation he had no symptoms.

SUMMARY

1. A case of penetrating wound of the right ventricle of the heart, with injury to the left anterior descending coronary vessels, treated by suture and ligation, is reported.

2. During the year after operation the patient showed no signs of myocardial infarction or diminution of cardiac reserve, and appears to be perfectly well.

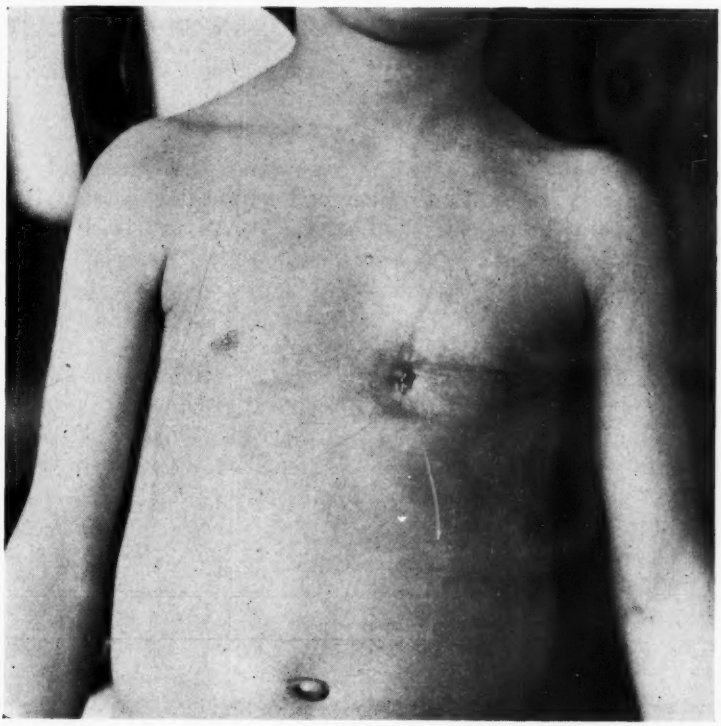

Fig. 5.—The patient two months after his operation.

3. Serial electrocardiographic tracings are presented. They do not show the changes due to anterior myocardial infarction as reported by Davenport in artificially produced coronary occlusion in dogs, or as reported by Olim and Hughes in their human patients.

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THE PROBLEM OF WOUND INFECTION IN THORACOPLASTY

Y. K. Wu, M.B., Ch.B., Peiping, China, and
Mario Besso Pianetto, M.D.
Rosario, Argentine Republic

WOUND infection is a serious complication in thoracoplasty for pulmonary tuberculosis. Alexander[1] estimated a mortality rate of 25 per cent in cases of deep infection, even with early proper treatment. In the instance of less severe infection, the delay between stages usually results in unsatisfactory collapse. Therefore, it is highly important for the thoracic surgeons to take every precaution to avoid the development of this complication.

At the Robert Koch Hospital, a review of the records of the two and one-half years between Jan. 1, 1940, and June 30, 1942, showed an incidence of wound infection of 11.8 per cent in 339 stages of thoracoplasty on 140 patients (Table I). In compiling the statistics in this

Table I

Incidence of Wound Infection in Thoracoplasty*

(Jan. 1, 1940, to June 30, 1942)

Thoracoplasty	No. of Operation	No. of Infection	Per Cent of Infection
First stage	140	14	10.
Second stage	132	17	12.9
Third stage	61	8	13.1
Fourth stage	2	0	0
Revision	4	1	25.
Total	339	40	11.8

*Four patients with thoracostomy for empyema and one patient with Monaldi drainage before the thoracoplasty were excluded from the statistics. Three of these five patients had infection of the thoracoplasty wounds.

series of cases, only instances in which frank pus was led out from the wounds were designated as infections. During the seven months from July 1, 1942, to Feb. 1, 1943, at the same institution, by following a routine to be described in this communication, we were able to carry out 120 consecutive stages of thoracoplasty on 55 patients without a single instance of wound infection (Table II). In the second series of thoracoplasties, not only the gross suppuration was unobserved but also the superficial infections and stitch abscesses were eliminated. We wish to report our experience in the latter group of cases to illustrate the fact that a perfect wound healing can be obtained by following

From the Robert Koch Hospital, Koch, Mo., and the Chest Service, Barnes Hospital, St. Louis.

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TABLE II

120 STAGES OF THORACOPLASTY WITHOUT WOUND INFECTION
(JULY 1, 1942, TO FEB. 1, 1943)

THORACOPLASTY	NO. OF OPERATION	NO. OF INFECTION
First stage	55	0
Second stage	47	0
Third stage	17	0
Revision	1	0
Total	120	0

the general principles of surgery, even under such an unfavorable situation as a thoracoplasty in a tuberculous patient.

Our precautionary measures against wound infection may be discussed under the following headings: (1) the elimination of contamination, (2) the operative procedure, (3) the postoperative care of the wound and (4) the general treatment of the patient.

THE ELIMINATION OF CONTAMINATION

Besides gross break in technique, there are two chief sources of contamination in a clean surgical incision: the patient's skin and the air in the operating room. The predominating bacteria in both instances are the staphylococci.[2] This organism was cultivated from thirty-two of the forty infected wounds of our first series. Our methods to minimize contamination from these two sources are as follows.

Skin Contamination.—Preparation of the patient's skin starts several days before the operation by cleansing the back with soap and water. In the evening before the operation, besides the usual shaving, the entire side of the chest is gently but thoroughly scrubbed with soap and water for five minutes. The scrubbing is repeated in the morning just before the patient is sent to the operating room. The skin of the operative field is treated with ether, 3.5 per cent tincture of iodine and alcohol. As soon as the skin and the subcutaneous tissue are incised, all the skin surfaces on both sides of the wound are covered with thick absorbent pads, which are held in place by the weight of the hemostats in the wound (Fig. 1). Sterile towels are then applied over the pads (Fig. 2). The use of the pads underneath the towels is very important, as a single layer of the towel is readily soaked through by blood or perspiration of the patient. At the end of the operation, the pads and towels are removed after the muscles and fascia are closed. The skin is treated with alcohol before the skin sutures are applied.

Air Contamination.—By radiating the air in the operating room with ultraviolet light, Hart[3] reduced his wound infection in thoracoplasty from 33 to 3.5 per cent and Overholt and Betts[4] improved their in-

fection rate from 13.8 to 6.53 per cent. In the absence of such special light installation, we use two mechanical means to minimize the air contamination. First, the wound is covered with towels in such a way that only the area necessary at certain stages of the operation is exposed to the air. The towels are changed at frequent intervals. Second,

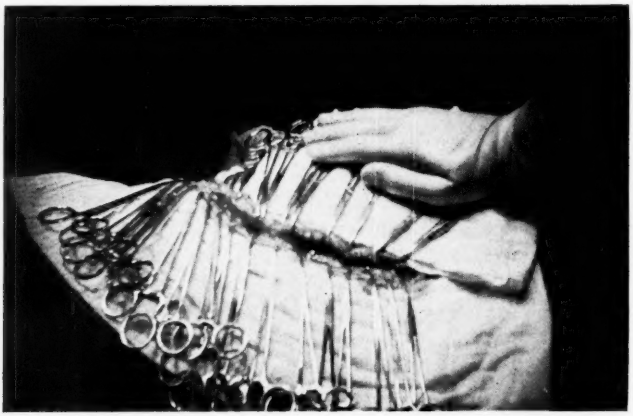

Fig. 1.—Skin surfaces on both sides of the incision are covered with thick absorbent pads, which are held in place by the hemostats.

Fig. 2.—The wound edges are covered with towels before the deep structures of the chest wall are opened.

at the end of the operation, the entire wound surface is irrigated with a large amount of warm normal saline. This latter procedure removes from the wound not only tissue debris and blood clots, but also an innumerable number of bacteria that may have been deposited from the air.

THE OPERATIVE PROCEDURE

Thoracoplasty is generally regarded as a very traumatizing operation. It is necessarily so because of the nature of the procedure. However, with care and sacrifice of unnecessary speeding, the injury to the soft tissue of the chest wall can be reduced to a minimum. The divided muscles are protected from the pulling and pressure of the retractors with moist pads and towels. Bruising of the periosteum and the intercostal muscles during the removal of the ribs should be avoided as much as possible. At the end of the operation all the loose soft tissue tags are removed before the wound is closed.

Closure of the wound is effected by a first layer of interrupted sutures of No. 0 chromic catgut for the muscles, a continuous suture of No. 000 chromic catgut for the subcutaneous fascia, and interrupted sutures of "A" silk for the skin. In using catgut, it is to be emphasized, as pointed by Howes,[5] that the smallest size of the suture material that gives enough strength to hold the wound should be chosen. The total amount of the suture material should be reduced to a minimum. For this reason, we secure hemostasis by including the clamped bleeding spots in the muscles and fascia in the closing sutures. It is only on rare occasions that an individual ligature is necessary. In the 120 incisions closed in this manner, demonstrable accumulation of sanguineous fluid in the wound occurred six times. The wounds healed well after removal of the fluid by aspiration. The closure of the skin with interrupted silk sutures gives perfect healing of the incision and has much to do with the reduction of the incidence of mild infections and stitch abscesses.

It is our general rule not to drain our thoracoplasty wounds. A drain was employed in only one out of our 120 operations. In our first series of 339 stages of thoracoplasty, drains were used 52 times. Of these 52 stages, infection took place in 14 wounds, giving an infection rate of 26.8 per cent. In the other 287 stages, where no drain was employed, 26 wounds became infected, giving an incidence of infection of 9 per cent. We are convinced, from these figures as well as from our general impression, that a drain is not necessary and should be omitted.

THE POSTOPERATIVE CARE OF THE WOUND

A pressure dressing by the use of mechanic's "waste" is applied at the end of the operation. A moderate pressure is effected to the wound to reduce tissue edema and to maintain good immobilization of the wound surface. In the case of a first stage thoracoplasty, it is usually not necessary to disturb the dressing until the sixth day, when the skin stitches are removed. However, in the subsequent stage operations, there is usually a considerable amount of serosanguineous drainage on the dressing and a change on the second or third day is required.

If a hematoma is found in the wound, the blood should be removed by aspiration. Reopening of the incision is only indicated when there is definite sign of infection.

GENERAL TREATMENT OF THE PATIENT

In order to secure satisfactory wound healing, attention to the general nutritional status of the patient is just as important as the local treatment of the wound. It is well known that anemia, hypoproteinemia, and hypovitaminosis predispose delay in wound healing and lowering of resistance against infection. Even though the nutritional status of the patient is normal before the operation, it is likely to be upset postoperatively. Therefore, in the immediate preoperative period, a high protein diet with additional vitamins should be prescribed. The same regime is to be resumed as soon as possible after the operation.

We give a blood transfusion of 500 c.c. at the end of each operation. This is considered to be necessary for replacement of the blood loss during the procedure.[6] It undoubtedly helps in promoting wound healing and hastening recovery of the patient.

SUMMARY AND CONCLUSIONS

Wound infection is a serious but rather frequent complication in thoracoplasty for pulmonary tuberculosis. However, by careful application of the general principles of surgery, its incidence can be reduced to a minimum. This is illustrated by a series of 120 stages of the operation without a single case of infection. The procedures employed in the prevention of infection of the wounds are discussed.

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WOUND HEALING AFTER THORACOPLASTY IN A TROPICAL HOSPITAL

RICHARD A. S. CORY, M.B., CH.B., M.R.C.S. (ENGLAND), L.R.C.P. (LONDON)
LIGUANEA, JAMAICA, BRIT. WEST INDIES

IN THE mind of the surgeon undertaking the treatment of pulmonary tuberculosis by collapse therapy, the dread of wound sepsis not uncommonly occupies a prominent place.

An extrapleural pneumothorax, for instance, may become an infected space, necessitating drainage and abandoning of what is, perhaps, the patient's last hope; while infection of a thoracoplasty wound, even if it does not end fatally, invariably lengthens the interval between stages to such an extent that subsequent operations often fail to produce satisfactory collapse. The sputum in these cases may remain positive, and the best that can then be hoped for is the rather problematical benefit to be derived from a revision operation.

Secondary sepsis in the subscapular wound may lead to involvement of tuberculous areas in lymphatic paths or lung, and may then bring about an added tuberculous infection, the eradication of which is often impossible. Under such circumstances a good-risk patient may be converted within a week or two into one for whom months of sepsis lie ahead.

The case may even prove fatal if the infection is of an overwhelming nature, and the patient's general condition at the outset not overly good.

Alexander[1] estimates that 25 per cent of the patients with acute wound infection with general disorganization of the wound after thoracoplasty will die before the sepsis can be overcome, even if treatment has been prompt and thorough; but he adds that the death rate will be higher than this if the diagnosis is delayed, or the treatment halfhearted.

It is generally believed that secondary sepsis in surgical wounds in the tropics is more commonly met with than it is in temperate climates, and I agree with this belief.

I am not aware, however, of any large series of figures to uphold this opinion, and I do not think that the matter has been thoroughly investigated.

From the King George V Jubilee Memorial Sanatorium, Liguanea.
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When infections occur, they are almost certainly of an exogenous nature and may possibly be the result of contamination by dust, from which very few of our tropical operating theatres are free.

The sound healing by primary intention of all surgical wounds, and particularly of the large serum and blood-filled spaces that result from thoracoplasty, is greatly to be desired, and if this end can be achieved with reasonable certainty by introducing a chemical substance into the wound at the time of operation, this addition to the technique would appear to warrant careful consideration.

The work of Deryl Hart[2, 3] at Duke University, N. C., on the use of ultraviolet light for sterilizing the air and tissues in the vicinity of an open surgical wound, is of supreme interest, but the equipment is likely to be costly and beyond the reach of many small hospitals, while the technique of preparation for the operation is on the elaborate side.

Allbritten, Flick, and Gibbon[4] have felt that the use of sulfanilamide in the chest after lobectomy and pneumonectomy definitely contributed toward lowering the incidence of pleural infection. Kent and Graham[5] produced very strong evidence in experiments on dogs to show that the use of the sulfa drugs introduced simultaneously into experimentally contaminated pleural cavities prevented the development of staphylococcal and streptococcal empyemas.

The last two writers, moreover, remarked that the wounds in the thoracic walls healed well in the animals in which the sulfa drugs had been used.

The work of Jensen, Johnsrud, and Nelson[6] on the results of implanting sulfanilamide directly into the wounds in cases of compound fracture lends additional support to the view that such a procedure is of great value.

While on a visit to Baltimore in Sept., 1941, I was struck by the free use of sulfadiazine in surgical wounds at the Johns Hopkins Hospital, and determined to try the effect of one of the sulfa drugs in my own patients with thoracoplasty wounds.

With this end in view, I obtained a letter of introduction from Arnold Rich of the Johns Hopkins Hospital to a member of the medical staff of E. R. Squibb & Sons.*

The two series of operations reported here run as closely parallel to one another as such series in actual hospital practice can be reasonably expected to do.

All the operations with the exception of some five or six were done by me, while the assistant was the same in all but about fifteen, and the instrument nurse the same in all but about twelve.

The operations were all done in the same operating theatre, and at the same time of day, between 2:00 P.M. and 4:00 P.M. This theatre is not air conditioned, and no fans are used. With but five exceptions,

*My sincere gratitude is due E. R. Squibb and Sons for the samples of sulfathiazole they very generously sent me for experimental use.

the patients in these series are colored Jamaicans, and a large percentage of them are Negroes.

The first ten or twelve patients were operated upon at intervals of six weeks between stages, but for the last two and one-half years this interval has been reduced to three weeks.

Essentials of surgical technique have not been varied, with the exception of that used in the anterior stages, where a complete silk technique is usually, but not invariably, employed and a detail relating to fascial closure, to be described later.

Preparation of the skin is done in the ward the night before operation with soap and hot water, followed by a rectified spirit wash, over the operation area.

The spirit wash is repeated the next day one hour before the patient comes to the theatre.

On the operating table, after the induction of anesthesia, the skin is again swabbed over with three lots of rectified spirit. A coat of two- and one-half per cent iodine tincture is then applied, a final wash off with spirit is given, and the patient is draped with the sterile towels and sheet to be used.

As will be seen from Tables I and II, drainage is very seldom employed, and the periostea of the ribs are generally formalinized.

TABLE I

WOUND HEALING IN 90 OPERATIONS BEFORE THE USE OF SULFATHIAZOLE

	NUMBER	PER CENT
No. of patients	33	
No. of stages	90	100
Primary healing	62	69
Stitches removed for serum but no sepsis	8	9
Superficial sepsis, a few stitches removed	15	17
Deeper sepsis remaining localized	2	2
Gross sepsis with disorganization of wound	3	3
Formalin used on periostea	81	90
Wounds drained at time of operation	5	6

Table I gives the results of ninety operations (not patients) done between April, 1940, and Feb., 1942, before sulfathiazole was first introduced, and Table II the result of ninety operations with the addition of sulfathiazole powdering of the wounds, from Feb., 1942, to April, 1943.

The five patients who died within a few days of operation have been excluded, owing to the impossibility of saying what type of healing would have occurred. Both series are otherwise consecutive.

The operation done is the modern type of thoracoplasty without Semb's apicolysis. No stage, with the exception of those on empyema

TABLE II

WOUND HEALING IN 90 OPERATIONS WITH SULFATHIAZOLE

	NUMBER	PER CENT
No. of patients	36	
No. of stages	90	100
Primary healing	84	93
Slight stickiness of stitch holes (no stitches needed removal)	5	6
Stitches removed for serum, but no sepsis	0	0
Superficial sepsis, a few stitches removed	0	0
Deeper sepsis remaining localized	1	1
Gross sepsis with disorganization of wound	0	0
Formalin used on periostea	61	68
Wounds drained at time of operation	0	0

or otherwise thickened pleura cases, consisted of more than three ribs, and in a number of second and third stage operations, only two ribs were removed.

The first six ribs, however, are removed as far as their cartilages, and the transverse processes of the vertebrae are also resected.

Owing to the difficulty of obtaining surgical supplies because of the war, it became necessary to use, catgut as little as possible, and all ligatures consist of silk, catgut being reserved for muscle sutures only.

I am conscious that this mixing of suture material in the same wound is looked upon as being an offense against surgical technique, but I have no occasion to regret it, and my results have convinced me that it makes no difference.

The major change in the operative technique mentioned above concerns the use of suture material for the fascial layer over the muscles.

Until one year ago this was done with catgut. The resultant wounds, however, were often bumpy even though well healed, and it was decided to use silk for this layer of sutures. The scars have since been appreciably smoother in appearance.

It may, of course, be argued that this change in practice has been the cause of the improved healing experienced during the last fourteen months, but personally I do not think that it has had much to do with it.

On the introduction of sulfathiazole there was some doubt as to whether it would be compatible with the formalin used on the rib periostea, but it was tried, and it would appear from the cases reported that no harm results from the use of the two chemicals in the same wound.

After the periostea have been formalinized, 5 Gm. of sterile powdered sulfathiazole are scattered over, and rubbed gently into, all ex-

posed raw areas, and the wound is then closed without drainage. No wounds are flushed out with saline or any other antiseptic.

The dressings applied are not touched again until the stitches are removed on the seventh day, when the wounds are strapped across, according to Alexander's technique, with several narrow strips of strapping to give them greater support after removal of the stitches.

The rule of "no change of the dressing until the seventh day," is broken only if there is enough ooze to make the dressings wet (about one case in every seven). There has been no case where any toxic effect from the drug was observed.

STUDY OF TABLES

Study of Tables I and II shows that in the presulfathiazole cases only 69 per cent of the wounds healed by primary intention, whereas after the use of sulfathiazole as a routine had been introduced, 93 per cent of the cases healed perfectly, and in over 98 per cent, the healing was satisfactory.

Before the use of sulfathiazole no fewer than 17 per cent of wounds had some superficial sepsis requiring the removal of one or more stitches, while a further 5 per cent developed severe sepsis, of which three were of the very grave wound-disorganizing type, one of these later becoming a tuberculous infection.

Since sulfathiazole has been routinely used, there have been no cases of superficial sepsis requiring the removal of even a single stitch, and in only five patients did very slight stickiness show around the stitch holes. There was, however, one case of deeper localized sepsis, but this occurred in the patient mentioned previously who had had gross sepsis before the use of sulfathiazole and who had developed a tuberculous wound infection. Under these circumstances, he is scarcely a fair test of the method, although he has to be included to make the cases consecutive.

CONCLUSION

In this hospital the use of sulfathiazole has greatly lessened our fear of infections, and even the patients feel that if they receive it, their stages will run through to schedule unhampered by sepsis and the consequent tedious and dangerous delays.

I feel that the results reported, even though they be relatively few, point very definitely to the beneficial effects of the local implantation of sulfathiazole into surgical wounds as an agent for the prevention of sepsis.

SUMMARY

A relatively parallel series of thoracoplasty operations is discussed with a view to comparing healing of the wounds before and after the use of powdered sterile sulfathiazole introduced into the tissues at operation.

Figures are produced which appear to show that wound healing in patients who have had sulfathiazole placed in their wounds at the time of operation is very much more satisfactory than in those who have not had it.

In eighty-nine out of ninety operations, not a single stitch had to be removed for even the most superficial sepsis, and the one exception occurred in a patient who had developed gross sepsis during an earlier operation before the use of sulfathiazole.

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TRANSPLEURAL, INTRATHORACIC LIGATION OF THE LEFT COMMON CAROTID ARTERY

The First Case Report

Frank P. Coleman,* M.D.
Columbia, S. C.

THE surgical approach to the common carotid artery has remained surprisingly constant. Heretofore, it has been ligated in the neck in spite of the presence of infection, malignancy, cervical hematomas, or secondary hemorrhage.

Ligation of this artery was first accomplished by John Abernethy,[1] in 1798. In 1941, Touroff[12] reported the first case of ligation of the first portion of the left subclavian artery through a transpleural approach. This author suggested that a similar attack could be carried out on the right side, ligating either the innominate artery or the right subclavian artery. A review of the available literature has failed to reveal any reference, suggestion, or application of a transpleural, intrathoracic approach to the ligation of the left common carotid artery.

The thoracic portion of the left common carotid artery measured, in various fresh cadaver specimens, from 3 to 5 cm. in length. In its course through the upper part of the superior mediastinum it runs upward and slightly laterally, terminating at the left sternoclavicular joint where the cervical portion begins. It arises immediately behind and to the left of the origin of the innominate artery from the aortic arch. Posteriorly, the thoracic portion of the left common carotid is in relationship from below upward with the trachea, the left recurrent laryngeal nerve, the esophagus, and the thoracic duct. The first portion of the left subclavian lies in a posterolateral position, but this relationship is not obvious until the mediastinal pleura has been divided and dissected anteriorly. The left innominate vein runs obliquely across the anterior aspect of the artery. Cardiac branches from the vagus nerve and sympathetic trunk descend vertically in front of the artery. Laterally, the artery is covered by the left pleura, and it is in relationship with the left phrenic and the left vagus nerves. By splitting the mediastinal pleura between the vagus and phrenic nerves the intrathoracic portion of the left common carotid artery can be easily exposed through its lateral relationships.

*Major, Medical Corps, U. S. Army.
From the Surgical Service, Brooke General Hospital, Fort Sam Houston, Texas.
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CASE REPORT

This 21-year-old white soldier entered the Brooke General Hospital, Jan. 13, 1943, for the treatment of a malignant melanoma of the neck. The local lesion had been excised elsewhere four months previously, only to recur in a lymph node at the angle of the left mandible. His general physical condition was excellent and an x-ray of the chest was negative for evidence of metastatic foci in the lungs.

Twelve days following his admission to this hospital a radical neck gland dissection on the left was performed under gas-oxygen-ether vapor anesthesia. The scar of the previous biopsy, the sternocleidomastoid muscle, the external and internal jugular veins, the external carotid artery, the contents of the submaxillary triangle, the inferior pole of the parotid gland, and intervening tissue and lymphatics were removed as a whole in the dissection. The pathologist reported, "a malignant melanoma completely replacing only the lymph node at the angle of the mandible. The other lymph nodes do not show the presence of metastasis."

On the sixteenth postoperative day, a brawny edema was present in the region of the submaxillary triangle and in the superior portion of the wound. On the twenty-fifth postoperative day, a large hematoma appeared in the neck at the junction of the transverse limb with the vertical limb of the scar. The hematoma was evacuated and promptly a massive hemorrhage amounting to 600 c.c. of blood resulted before the bleeding could be controlled by digital compression. Under local anesthesia, the wound was opened wide. There was a gross ulceration of the common carotid artery at the site where the external carotid artery had been ligated. It was not possible to ligate the common carotid artery proximal to the site of bleeding. There was a woody and dense formation of scar tissue resulting from the presence of a diffuse inflammatory process. A mass ligature of double chromic No. 2 catgut was passed on a needle beneath the common carotid artery, but the suture could not be tied securely enough to control the bleeding for fear of cutting through the existing damaged arterial wall. Two additional mass sutures of chromic No. 2 catgut were then placed immediately about the bleeding point and the hemorrhage appeared to be temporarily under control. At the onset of the operation, the blood pressure was 100 mm. of mercury, systolic, and 50 mm. of mercury, diastolic. The pulse rate was 116 per minute. Two days prior to the hemorrhage, the hemoglobin was 80 per cent; white blood cell count, 9,750; red blood cell count, 4,250,000. A 500 c.c. blood transfusion was given during the operation and on no occasion did the blood pressure fall below 100 mm. of mercury, systolic. These preoperative findings indicate an optimum general condition for the occlusion of the common carotid artery. The initial ligation was complicated by the develop-

ment of a husky voice and the appearance of a Horner's syndrome on the first postoperative day. These neurologic manifestations were attributed to the incorporation of the sympathetic trunk and the vagus nerve in the mass ligature.

On the second postoperative day, it was obvious that this occlusion of the common carotid artery would not be sufficient to prevent a fatal outcome. Pulsations in the artery below the thyroid cartilage were still present. The infection had descended to a level of the clavicle and this rendered an approach to the common carotid artery at a lower level in the neck impractical. The only method which seemed anatomically and surgically sound was a ligation of the artery in an area free of infection. The intrathoracic portion of the left common carotid artery fulfilled these requirements.

Seven days following the mass ligation, the common carotid artery was observed to have strong and full pulsations. The operating room was made ready; however, a second massive hemorrhage of about 500 c.c. of blood occurred before the patient could be transported to the surgical theater (Fig. 1). Digital pressure controlled the bleeding which was maintained during the induction of gas-oxygen-ether vapor anesthesia and until the following procedure could be carried out.

Surgical Technique.—A transverse skin incision was made over the second intercostal space beginning at the lateral border of the sternum on the left and extending to the anterior axillary line. This approach is similar to the one recommended by Gross[6] for the ligation of a patent ductus arteriosus and by Touroff,[12] who used the approach in ligating the subclavian artery by way of a transpleural incision. The pectoralis major and minor muscles were divided in a line with their fibers and the left pleural cavity was entered through the second intercostal space (Fig. 2). The internal mammary vessels were doubly ligated and divided. It was not necessary to divide the rib cartilage. A rib spreader was introduced and the upper lobe of the lung was covered by a moist pack and retracted downward until the superior aspect of the hilus of the lung was visible (Fig. 3). The mediastinal pleura was opened over the arch of the aorta and the incision extended upward for a distance of three inches midway between the phrenic nerve anterior and the vagus nerve posterior. On dissecting the posterior leaf of the mediastinal pleura with the vagus nerve, the subclavian artery was easily exposed and identified. The anterior leaf of the mediastinal pleura was now mobilized with the phrenic nerve and the left common carotid artery came readily into view (Fig. 4). The intrathoracic portion of the common carotid artery was 4 cm. in length. Two ligatures of No. 8 braided silk were tied 1 cm. apart about the left common carotid artery 2 cm. from the arch of the aorta. It was observed during the development of the carotid artery that

ligation of the innominate artery could also be carried out without difficulty through this approach. The mediastinal pleura was closed with interrupted sutures of fine silk. The pleural cavity was washed with physiologic saline solution. The lung was re-expanded by positive pressure and the pleural cavity and wound were closed without drainage.

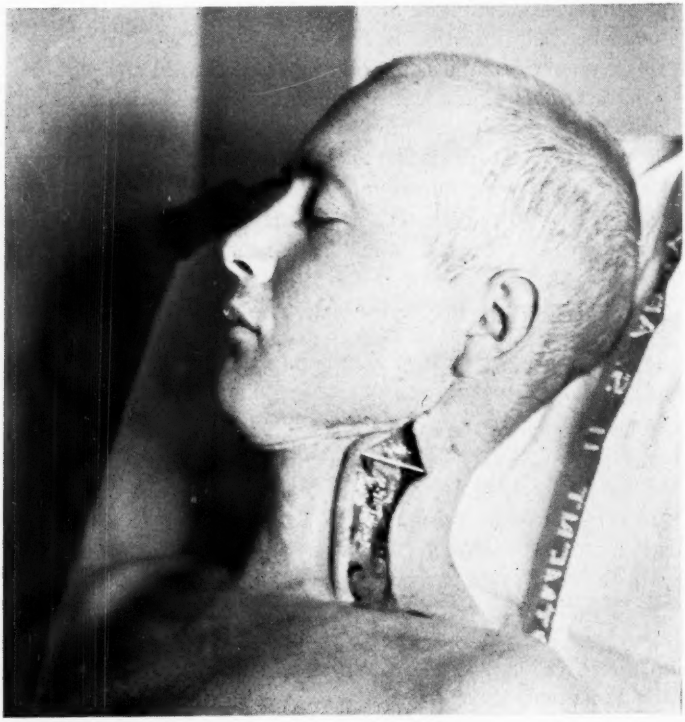

Fig. 1.—Extensive wound infection; appearance prior to second massive hemorrhage.

A 500 c.c. citrated blood transfusion was given preoperatively and during the course of the operation an additional 500 c.c. of blood was given. The blood pressure was always maintained at a level above 100 mm. of mercury, systolic, and 65 mm. of mercury, diastolic, during the operation. At the time of occluding the common carotid artery, the blood pressure was 126 mm. of mercury, systolic, and 70 mm. of mercury, diastolic. Two days prior to the intrathoracic ligation, the hemoglobin was 65 per cent; the red blood cell count was 3,080,000; and the white blood cell count was 14,300. The patient was in good general condition on the second occasion for occluding the artery and no neurologic symptoms were evident either at the time of the operation or thereafter.

The left pleural cavity was aspirated on two occasions, which yielded 100 c.c. of a serosanguineous fluid and 650 c.c. of the same type of fluid on the first and third postoperative days, respectively. There was no further bleeding from the wound and the carotid artery pulsation has

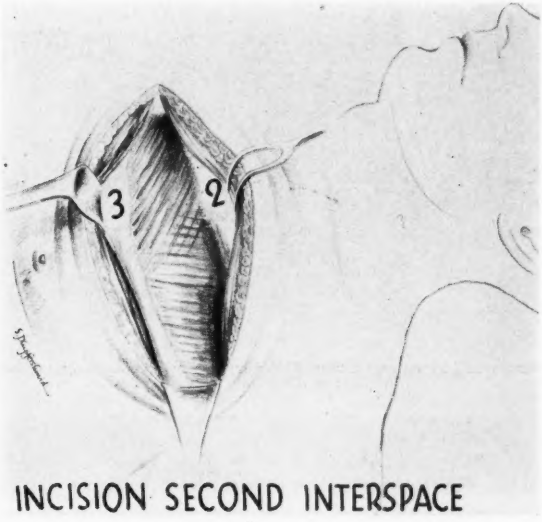

Fig. 2.—Surgical approach, incision in second interspace.

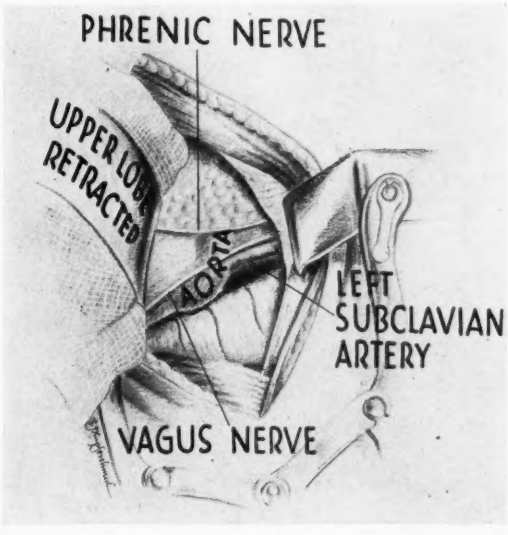

Fig. 3.—Anatomic landmarks on retraction of upper lobe downward.

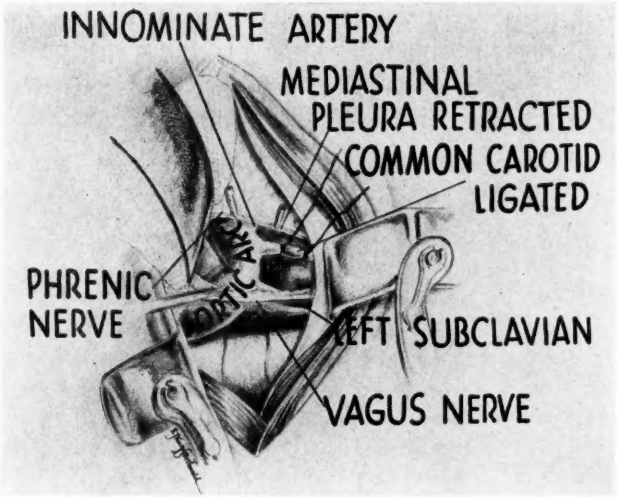

Fig. 4.—Exposure of left common carotid artery; note adaptability of subclavian and innominate arteries to ligation.

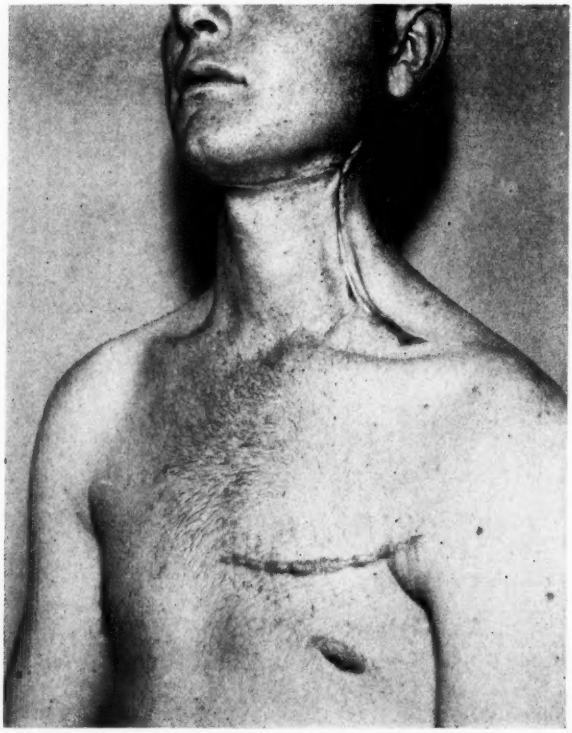

Fig. 5.—Forty-two days postoperative.

remained persistently absent in the neck. On the third postoperative day, a left lower lobe pneumonia was detected, which cleared rapidly in ten days with sulfadiazine therapy. He had entirely recovered on the twenty-first postoperative day, with the exception of the neck wound where healing was completed forty-two days after the second ligation of the left common carotid artery (Fig. 5). At this time, the larynx was normal; funduscopic examination revealed the eye grounds to be normal; the Horner's syndrome had improved; and there was no evidence of a recurrence of the initial lesion.

DISCUSSION

Anatomically, the common carotid arteries are surgically accessible in the neck; however, below the omohyoid muscles exposure is more difficult and dangerous in that the carotid arteries here are overlapped by the great venous trunks at the base of the cervical region.[2, 4, 8] In exposing the innominate artery[8] it has been suggested that a portion of the manubrium might need to be resected in ligating this vessel. These anatomic findings are surmountable by an approach through the neck, but it is not difficult to entertain circumstances which will not only render ligation of the infraomohyoid portion of the common carotid arteries difficult but impossible.

The development of an infection within the region of the carotid vessels either postoperatively or spontaneously is associated with the everlasting fear of an exsanguinating hemorrhage. Thrombosis of the artery does not often result, but the infection invades the arterial wall and the inevitable hemorrhage occurs. In the management of 113 consecutive patients with cancer of the gums, hemorrhage was a complicating factor in 23 patients and the cause of death in 6 patients.[11] Ligation of the common carotid artery was performed 20 times at the head and neck tumour clinic of the Memorial Hospital, New York, from 1926 to 1937; and Watson and Silverstone[13] cite actual or threatened hemorrhage as the indication for the occlusion of this vessel in 13 cases. In 12 of these 13 cases an associated infection and sloughing of the wound played a predominant role. Eleven cases in the series of 20 had a neck gland dissection and in this group 7 developed an actual secondary hemorrhage either from an inflammation of the arterial wall or from sloughing of the stump of the previously ligated carotid artery. Greenfield[5] reviewed the literature for a period of twenty years prior to 1933, and was able to report 31 cases where a serious hemorrhage from the neck was a result of infection. Hemorrhage from the carotid arteries either postoperatively or otherwise in the absence of a wound is primarily a manifestation of an infection. Ligation of an artery in the presence of inflammation will likely be followed by a secondary hemorrhage. In neck infections where access to some healthy area of the artery cannot be obtained without traversing a septic field, the surgical approach described seems safe and surgically sound.

Recent experiences of neurosurgeons[3, 9] in the management of aneurysms of the internal carotid by the ligation of this vessel disclose that, with proper preparation and selection of cases, the mortality rate will not exceed 8 per cent and the cerebral complications will be infrequent. The application of ligation and extirpation to either an aneurysm or an arteriovenous fistula involving the common carotid should offer a commensurate prognosis. The most common site for an aneurysm of the common carotid is its origin or bifurcation. In the region of the bifurcation of the common carotid the jugular vein may become blended with the carotid sheath and eventually with the aneurysmal sac. Such a complication would add to the difficulties of either extirpation or ligation of the artery within the neck. In either aneurysms or arteriovenous fistulas of the inferior one-third of the common carotid, and of the innominate artery, the intrathoracic approach may find a field of usefulness.

In the present war, injuries of the common carotid arteries inflicted by flying missiles or debris and by stab wounds will not be infrequent. Experiences in World War I showed that such an injury may result in death immediately from exsanguination, the formation of a large hematoma in the cervical region, or an arteriovenous fistula. The difficulties attending the approach to the common carotids in the presence of a massive hematoma of the neck, and the high incidence of fatal cerebral accidents accompanying early ligation, led Makins[10] to believe that it was never wise to operate in such cases. Homans[7] did not believe that operation should be undertaken in injuries of the common carotid artery in the absence of dangerous extension of the hematoma and in the absence of external hemorrhage. This author also pointed out the danger of a secondary hemorrhage resulting from ligation of the common carotid in the presence of an infection. The transpleural, intrathoracic approach seems to offer a field suitable to the ligation of the common carotid arteries far removed from the site of the primary injury, an area free from contamination, and a method of side-stepping the dangers and hazards attached to an approach through a hematoma in the cervical region. One of the primary principles in dealing with a wound of an artery is the exposure of the vessel proximal to the site of the injury.

SUMMARY AND CONCLUSIONS

1. The reported case appears to be the only attempt recorded in the literature to ligate the left common carotid artery through a transpleural, intrathoracic approach.

2. The ability of patients to withstand a thoracotomy without ill effects under the most trying circumstances is now well established.

3. The surgical technique of this approach has been discussed and illustrated in detail. It can be rapidly and safely executed without a residual deformity of the bony thorax.

4. It appears as if the transpleural, intrathoracic approach would find a field of usefulness in the management of an actual or an impending hemorrhage secondary to a diffuse neck infection, in arteriovenous fistulas and aneurysms situated low in the cervical region, and in traumatic injuries of the neck accompanied by massive hematomas. In such incidences it is suggested that this approach may be used alone, or in combination with some other suitable procedure in the cervical region.

5. It is anatomically feasible to use a left-sided approach in ligating the first part of the left subclavian, the left common carotid, and the innominate artery. Through a right-sided approach it is possible to ligate the innominate, the right common carotid, and the first part of the right subclavian artery.

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BRONCHOSCOPY IN PULMONARY TUBERCULOSIS

MILTON SILLS LLOYD, M.D., AND JOSEPH A. BUDETTI, M.D.*
NEW YORK, N. Y.

THE FUTURE OF BRONCHOSCOPY†

THE greatest exponent of bronchoscopy whom the world has known, or is likely to know, Chevalier Jackson, has repeatedly asserted, "There are three ways of examining the lung. We may look at it from the outside by inspection, palpation, and auscultation; we may look through it by means of the x-ray; and we may look at it from the inside by means of the bronchoscope." This immediately assigns bronchoscopy to its rightful place as a specialized method for the diagnosis and treatment of diseases of the lungs and, inferentially, requires that it should be placed in the hands of the lung specialist for this purpose.

The above statements are self-evident facts, hardly admitting the necessity of supporting proof. Still, because of its origin, bronchoscopy remains, in most places, fettered to the nose and throat department. The lung specialist acknowledges gratefully the debt which he owes the otolaryngologist for his pioneer work in intrapulmonary pathology. But he does not admit that this automatically permits the otolaryngologist to affix to the end of his eye, ear, nose, and throat field the specialty of lungs. The training of the nose and throat specialist is at the wrong end of the bronchoscope and he has no essential interest in the use of his instruments. A friend of mine, a nose and throat specialist, told me, "The bronchoscope is my plaything." Another said that he kept his bronchoscope with his fishing tackle and used it for the same purpose and with about the same frequency. Each of these men was in charge, at the time, of the bronchoscopic service in a large hospital in New York. At the same time, I, who found bronchoscopy an indispensable part of my training, was denied bronchoscopic privileges in both those institutions because I was a lung specialist.

The fallacy of this establishment, still found in most hospitals, is apparent. The nose and throat specialist, having acquired a bronchoscope, is dependent upon the medical man to tell him when to use it and, apart from the removal of foreign bodies, he endeavors only, as one of them has put it, to establish "a high batting average in confirm-

*First Lieutenant, Medical Corps, U. S. Army.

†The opinions expressed in the section on The Future of Bronchoscopy are those of the senior author.

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ing the diagnosis." As a consequence, both parties are prevented from using the instrument as an independent means of investigation. During the past ten years, I have spent much of my time in teaching bronchoscopy to nose and throat specialists. They have been, without exception, men of the highest mental and professional caliber. I have still, however, to learn of one of them who has been able to exploit this training in private practice.

In the field of thoracic surgery, the same difficulties immediately become apparent. No one can tell as well as the surgeon himself when a lung abscess requires external rather than endoscopic drainage. No one else will give the bronchiectatic patient such intelligent and careful preoperative care and no one else can evaluate so accurately the significance of endoscopic findings in the cancer case. In the field of pulmonary tuberculosis, the situation becomes hopeless. The nose and throat specialist does not understand pulmonary pathology. He is not familiar with the interpretation of x-ray films of the chest. His observations, therefore, are limited to the familiar ulcer and cicatricial stenosis, one of which he finds it necessary to cauterize and the other to dilate.

The time has now arrived when the bronchoscope must be given its place of equality with the x-ray, even in the treatment of pulmonary tuberculosis. The phthisiologist does not use the x-ray to treat pulmonary tuberculosis, but he cannot treat the disease without it. The same may be said of the bronchoscope.

The great iniquity of the present setup is not seen in the large centers where bronchoscopy is done, but in suburban and rural districts where it is not. The several methods used in the diagnosis and treatment of lung disease remain divided among many workers. As a result, the same confusion exists in the thoracic surgical field as would exist in urology if that field were suddenly divided into diagnosis, radiography, bladder irrigations, cystoscopy, and surgery, each part under a different man. To get the urologist's impression of the situation, it is only necessary to ask him what would happen to his practice if cystoscopy were taken away from him. The outcome in relation to the specialty of lung diseases is that, in large centers, where adequate help can be found, four or five men must be called upon to do the work of one. In small centers, the work is either done badly or the community is left with no service whatsoever in this field. I have seen bronchoscopy used in the treatment of the following conditions, because of inadequate knowledge on the part of the bronchoscopist: pleurisy with effusion, empyema, aneurysm of the aorta, spontaneous pneumothorax, and heart failure. I have been called upon to travel a thousand miles to perform a bronchoscopy in a prosperous town of 60,000 people. All this, of course, is ridiculous, since the technique of bronchoscopy has been perfected for a period of more than twenty-five years.

It is becoming increasingly apparent that the lung specialist can never consider himself adequately trained until he has mastered the art of bronchoscopy and, likewise, that bronchoscopy will never render its maximum service to medicine until it is embraced by the specialty to which it rightfully belongs.

THE CONTRIBUTION OF BRONCHOSCOPY TO THE MANAGEMENT OF COLLAPSE THERAPY

This study is based upon the objective findings in 475 bronchoscopies. of these, 385 were performed at Otisville Sanatorium and 90 on private patients.

None of these examinations were made as a routine procedure. The growing alertness of medical staffs to the indications for bronchoscopy is shown by the fact that abnormal findings were encountered in practically all the cases.

Physical findings observed bronchoscopically in pulmonary tuberculosis may be either a direct result of the disease itself, or, as a consequence of collapse procedures, these changes may be modified or new ones may appear. The subject is, therefore, treated under two heads:

Part I: Abnormalities resulting from the disease itself and

Part II: Abnormalities or modifications of existing abnormalities resulting from collapse therapy.

PART I

TYPICAL LESIONS; DIAGNOSTIC AND PROGNOSTIC VALUES

Neither suitable terminology nor suitable classification has been developed to answer the requirements of this phase of pulmonary pathology. It should be emphasized, however, that the term tracheobronchial tuberculosis[1] should not be used in referring to intrabronchial lesions, since it is already in common use with a definitely accepted meaning.

The following fundamental concepts have been developed and are adhered to throughout this communication in the interpretation of our bronchoscopic observations:

a. A lesion in the parenchyma of the lung always results in changes in the mucous membrane of the draining bronchus.
b. The lesion in the bronchus is of the same character as the lesion in the lung.[2]
c. The bronchial lesion is a direct extension into the visible airways from the original site of invasion (lung parenchyma or lymph gland).
d. Changes in the pulmonary lesion are constantly signalized by changes in the bronchial lesion.
e. Bronchoscopy, therefore, affords an avenue for direct study of the character and progress of a tuberculous lesion in the lung.

Many of these changes are neither gross nor striking in appearance and considerable experience is necessary to be able to detect and evaluate them. They may be classified under the following groups:

I. Nonulcerative lesions:
   1. Acute tuberculous bronchitis
   2. Chronic tuberculous bronchitis:
      (a) granulomatous variety
      (b) infiltrative variety
      (c) productive variety

II. Ulcerative lesions:
   1. Extrabronchial origin
   2. Intrabronchial origin

All of these are secondary to pre-existing pulmonary or glandular foci. They result from an extension either along or through the bronchial wall or as a "reaction" in the mucous membrane to the stimulus of an irritating discharge.

Nonulcerative Lesions.—The acute reaction is rarely seen because in this stage of pulmonary tuberculosis there is no indication for bronchoscopy, except for differential diagnosis. It consists of an injected pallor similar to that seen in the larynx. There is a slight degree of swelling and edema, particularly at the mouth of the bronchus leading to the involved area. Scattered blood vessels are visible to the naked eye (Case 1).

Chronic tuberculous bronchitis is by far the most important of the changes found. It is the lesion most frequently seen and, in its different shadings, has a definite significance in reference to the pulmonary pathology. The fundamental change is an infiltration of the normal mucous membrane by inflammatory tissue. There is an accompanying stream of milky gray pus representing the liquefied end product of the caseous process, in which tubercle bacilli can nearly always be demonstrated. This discharge must be carefully distinguished from the bronchial mucus in which tubercle bacilli are not found, until it is mixed with pus. In the draining bronchus, they frequently remain quite distinct from each other. Where the pulmonary lesion is inactive, the milky discharge is not seen, but absence of discharge cannot be interpreted as an absence of activity.

Variations in the chronic reaction constantly signalize the progression or regression of the pulmonary disease. They may be characterized as granulomatous, infiltrative, and productive. In the granulomatous variety, the surface of the mucous membrane is covered with fine mammillations of granulation tissue. Different degrees of edema are seen in these elevations. The infiltrative variety exhibits a much greater thickening of the mucous membrane. There is invasion of both mucosa and submucosa by inflammatory cells with an actual reduction

in the size of the bronchial lumen. The quantity of purulent and mucous discharge is increased. When associated with collapse therapy, cyanosis and congestion are added to the picture and the lumen of a lobar bronchus may be reduced to toothpick size. The productive variety of bronchial lesion is a substitute mucous membrane consisting of flattened epithelial cells mixed with fibrous tissue. It is smooth and pale pinkish yellow in color. As stated above, there may be mucus production, but the thin irritating drainage is not present. In such cases, although the sputum may yield an occasional low Gaffky count, it is difficult to demonstrate bacilli by any method.

The taking of smears through the bronchoscope is a practice advocated by some. Secretions are easily carried on the instrument from the trachea into either bronchus and are easily displaced from one bronchus to the other. Since the course of treatment is frequently determined by bronchoscopy, the most rigid accuracy must be observed. It is best achieved by using a very fine suction tube to collect the specimen within the lumen of the draining bronchus. Cases 2 to 6 will illustrate the clinical value of these observations.

At this point, mention should be made of the so-called allergic tuberculous bronchitis. Brunn and his associates[3] observed such changes. Their existence will be disputed by many. In some instances, however, either alone or in the midst of a chronic lesion, there appears an elevation in which the edema is so marked that it could almost be described as a blister (Case 7). Brunn suspected that such lesions might produce a "very efficient valve mechanism." In one of our cases, this valve was actually seen in operation (Case 8).

These elevations appear and disappear in the course of the disease. They may produce complete or partial obstruction and bear an important relation to changes in the lung distal to their position in the bronchus.

From the above observations, it will be seen that the presence of the chronic reaction in, and of discharge from, the draining bronchus is the most accurate index in our possession as to the source of positive sputum and the site of active disease. Bronchoscopy, therefore, is an indispensable supplement to x-ray examination in the settlement of these important and difficult problems.

Ulcerative Lesions.—Ulcerative lesions may be of extrabronchial or intrabronchial origin. The former are the result of rupture of a caseating lymph node into the airways. The latter represent an extension along the wall of the bronchus from a similar lesion in the lung. Since different methods of treatment must be used for the two conditions, the recognition of their etiology is manifestly important. Table I shows a comparison in characteristics between them. Many of these points can be recognized only by means of the bronchoscope.

TABLE I

ULCERATIVE LESIONS

Extrabronchial origin due to rupture of caseating lymph agglomeration through a bronchial wall	Intrabronchial origin due to extension of pulmonary lesion along bronchial wall
1. Positive sputum occurs late with low Gaffky count	1. Positive sputum precedes lesion with high Gaffky count
2. Localized perforation, destruction, or distortion of cartilage	2. Destruction of cartilage rare or late
3. May occur at any point, generally lower lobe, or stem bronchi	3. Extension upward from mouth of draining bronchus, generally upper lobe
4. Obstruction due to collapse of wall and intrabronchial lymph granuloma	4. Obstruction due to edema, false membrane, or granuloma
5. Granulomas of healing may occur as isolated "warts" in larger branches	5. Healing is accompanied by contraction of partial or complete cicatricial ring
6. Large lesions leave craters lined by fibrous tissue and replacement mucous membrane	6. Extensive lesions may result in complete closure by organized cicatricial floor

Ornstein and Epstein[4] report cases in which the disease is "predominantly bronchial." These, of course, are of extrabronchial origin. Pneumothorax cannot be used in the treatment of these lesions. It can do nothing more than further impair drainage in a situation which is already bad. In fact, the accumulated tuberculotoxins may be aspirated into the lung with the precipitation of a tuberculous pneumonia (Case 9).

The usual clinical course is much more favorable. In many instances, there is pressure upon a bronchus without perforation (Case 10). Or perforation may take place after a number of years (Case 11).

If perforation takes place without parenchymal involvement, the prognosis is relatively good. In two of our cases (Cases 12 and 13) we were able to observe bronchoscopically the healing of perforations from caseating lymph glands coincident with a return of negative sputum.

Ruptured glands, even when unsuspected, may also explain a positive sputum where treatment has been used unsuccessfully, or with unexplained success (Cases 14 and 15).

In addition to direct perforation, the lesion may make its first appearance in the bronchus as an inflammatory granuloma (Cases 16 and 17).

The advisability of biopsy for diagnosis is brought into question by the history of the latter case.

Early destruction of cartilage and collapse of the bronchial wall is a characteristic finding. This may cause the ulcer to be transmitted to the opposite side, giving the "bread and butter" appearance of a contact ulcer when the lumen is widened by the descending instrument (Case 18).

Ulcers of intrabronchial origin (Case 19) are an extension of the pulmonary lesion into the visible airways. They appear at the mouth of the draining bronchus and extend upward with the stream of infected discharge. According to Auerbach[5] these erosions are continuous in the anatomic specimen. This is also true bronchoscopically, although islands of healthy mucous membrane sometimes appear within the ulcerated area (Case 20). Myerson[6] and Kernan[7] describe lesions opposite to, or remote from, the draining bronchus. In our series, these transplants have not been seen.

Healing of intrabronchial ulcers is conditioned upon the development of a negative sputum. They will then show a progressive improvement paralleling that of the lung. Dead tissue is either liquefied or sloughed out; the defect is filled by granulations and finally bridged by fibrous tissue cells, together with a substitute mucous membrane. In superficial ulcers, only the mucous membrane and submucosa are destroyed. Invasion of cartilage is seen in terminal cases.

Healing is accompanied by some degree of bronchostenosis. If the ulcer is superficial and longitudinally distributed, it may be slight (Case 20). A bronchus which is draining never closes (Case 21). As soon, however, as the discharge is reduced to a point where it can be removed by local circulation (blood and lymph) it may close. Its fate depends upon the depth of the ulcer and whether the mucous membrane has been circumferentially destroyed. Given these three essentials, (a) lack of drainage through the bronchus, (b) an ulcer which reaches the submucosa, and (c) a distribution which rings the bronchial lumen, stenosis must take place and closure may be complete whether the cavity is open, encapsulated, or healed (Case 22). Pneumothorax or other form of collapse therapy may hasten this denouement by encouraging healing in the lung and by causing some mechanical reduction in the bronchial lumen.

Careful distinction must be made between occlusion and closure. Bronchi frequently appear to be closed but a few moments' observation or a cough will reveal the stream of watery purulent discharge which filters through the obstruction.

PART II

COMPLICATIONS AND SEQUELAE OF COLLAPSE PROCEDURES

Three methods of collapse therapy are applied to the lungs in the treatment of pulmonary tuberculosis: pneumothorax, thoracoplasty, and diaphragmatic paralysis. It is not within the province of this communication to discuss the surgical aspects of any of these methods. They have one common result—they all impair pulmonary drainage, although pneumothorax is by far the worst offender. Otherwise, their effects upon the lung differ greatly. Some of these effects may be seen bronchoscopically. They may be studied under the headings of Mechanical and Physiologic Changes.

Mechanical Changes.—Torsions and distortions of the bronchi vary in pneumothorax, depending upon the number and disposition of adhesions. In cases where the pleura is free, the lung falls into the posterior costal gutter, pulling the stem bronchus backward and rotating it away from the mediastinum; that is, clockwise on the right, counterclockwise on the left. The reduction in the volume of the lung is shared by the bronchi and this, combined with the torsion, may cause an inward bulging of the free end of the cartilaginous rings, resulting in wheeze or dyspnea, or both (Fig. 17).

Emphysema markedly accentuates this change and the posterior wall may obstruct the bronchus to such an extent that it produces a loud musical sound during cough. It may completely obstruct vision with the bronchoscope in situ. In some instances (Case 23) even the stem bronchi may be almost completely occluded by this mechanism.

The upper lobe is generally the seat of the greater amount of disease. Its increased weight when collapsed may cause it, on rare occasions, to fall downward behind the partially collapsed lower lobe. In one of our cases (Case 24) this displacement caused the mouth of the left upper lobe branch to appear bronchoscopically below that of the lower lobe branch.

The belief held by some that pneumothorax kinks the bronchi is fallacious. On the contrary, pneumothorax tends to bring the branch bronchi more nearly into alignment with the parent trunks. This frequently affords an excellent view of the upper lobe branches (Case 25). Adhesions may prevent pneumothorax from producing this effect and, under certain circumstances, kinks of the bronchi may result.

Under thoracoplasty, however, the apex is not dropped unless decollation is performed, and even then, it will partially recover its normal position. Ordinarily, the lateral part of the upper lobe is displaced downward and toward the mediastinum. This produces kinking in the majority of cases. In one instance (Case 26) this was so marked that the roof of the upper lobe bronchus was buckled in and appeared to be fractured.

The third method, diaphragmatic paralysis, causes a shortening of the bronchi from below upward. It is not enough, however, to satisfy the native elasticity of the lung and very little deformity results. The most notable change bronchoscopically is immobility.

Physiologic Changes.—For reasons that need not be discussed here, both ventilation and circulation are decreased in the collapsed lung. These changes are seen particularly in the lower lobes, where a remarkable degree of congestion and cyanosis may be found. The cyanosis is heightened by impaired drainage. In some instances, the entire bronchial tree may be filled by a stagnant discharge so that little or no oxygen can reach the lobe. The mucous membrane reaches a very unhealthy condition under such circumstances. It is thickened, deeply

cyanotic, glazed in appearance, and doughy to the touch of an instrument (Fig. 21). With time, these changes creep upward until they meet the altered mucous membrane of the draining bronchus. An ulcer frequently results from this unhealthy union. We have seen this take place in four of our cases. It can be prevented by partial re-expansion to permit drainage of the lower lobe (Case 27).

In a parallel case, where the lung was not re-expanded (Case 28), the patient ran a progressively downhill course. The sputum, at the same time, was nearly always negative because of failure of drainage of the involved lung. It became highly positive coincidentally with the development of a large cavity in the opposite lung.

The term atelectasis is applied rather loosely, in a clinical sense, to all conditions in which there is an x-ray shadow corresponding to a lung or an anatomic part of a lung. To the bronchoscopist, however, this x-ray appearance may represent several different clinical states. There may be (a) a healed pulmonary lesion with the draining bronchus closed by healthy scar (Case 22), (b) a healed pulmonary lesion where the bronchus remains open and the mucous membrane is intact (Case 25), (c) an obstruction to the bronchus in the absence of pneumothorax (Case 30), or (d) an obstruction to the bronchus in the presence of pneumothorax (Cases 15 and 28). Atelectasis is a valuable word. It signifies a demonstrable bronchial obstruction which, if removed, permits at least temporary reaeration. It should be reserved to describe this condition. Pneumothorax as an impediment to pulmonary drainage helps to cause atelectasis.

Atelectasis, as pointed out by Rafferty and Shields,[8] is attended by a train of serious complications. Pneumothorax, therefore, should be used only after due consideration of the ability of the lung to maintain drainage. It is unfair to this method, however, to say that it is contraindicated where there is moderate ulceration which progresses or does not regress. A similar opinion was once held by Alexander[9] in respect to thoracoplasty. He was, however, one of the first to revise it.[10] The immediate effect of pneumothorax may be to cut down drainage to such an extent as to more than compensate for the resultant impairment. In this way, pneumothorax, as well as thoracoplasty, may be the principal agent in bringing about the healing of an ulcer (Case 29). If drainage fails under pneumothorax, bronchoscopy cannot be used as a substitute for the natural mechanism.[11]

TREATMENT

Much of the literature on this subject is devoted to bronchoscopic treatment, consisting of cauterization of ulcers and dilatation of strictures.[7, 12-21] In view of the underlying pathology, it is difficult to see how either of these procedures could be of much value. In our series ulcers of extrabronchial origin showed a marked tendency to heal

except where there was progressive parenchymal involvement. This combination sets up a vicious circle which responds poorly to any form of treatment. The picture further deteriorates when complicated, as it frequently is, by a bronchial infection of pyogenic origin (Case 9). As in the case of tuberculous laryngitis, ulcers of intrabronchial origin follow the course of the parenchymal lesion. In some of our cases (Case 29) healing took place within a period of two months without bronchoscopic treatment. In no case was cauterization of any kind demonstrated to be of value.

Bronchostenosis must be treated with an equal degree of caution. We have never seen a permanent increase in lumen brought about by dilatation. On the other hand, pain, hemoptysis, increased sputum, and even the reappearance of tubercle bacilli and ulcers have followed instrumental stretching of a healthy scar. In one instance (Case 30), tubercle bacilli appeared in the sputum for the first time after the dilatation of a stricture formerly thought to be congenital. Like every other form of therapy, bronchoscopy should be directed with, and not against, the natural processes of repair.

Our attempts to influence the closure of bronchi by endoscopic interference have been equally unsuccessful since changes in the bronchial lumen are dependent upon factors beyond the reach of the bronchoscope. (See Case 21.) If these factors are changed by other methods of therapy, the effect upon the bronchus may be immediate and startling. This is illustrated by Case 31.

CASE REPORTS

Case 1.—J. K., a white man, 24 years old, was suffering from hemoptysis following second degree sunburn. Sputum was negative for tubercle bacilli. Bronchoscopy showed acute reaction. Specimen from the left upper lobe branch was Gaffky I. (Fig. 1.)

Case 2.—F. B. was a white woman, 36 years old, whose onset occurred Sept., 1937, in the right upper lobe. March, 1938, there was hemoptysis of one quart. Sputum was negative. Right pneumothorax was induced immediately. Satisfactory progress was shown up to Oct., 1940, when sputum became positive and increased. X-ray showed a questionable lesion in the left upper lobe. Nov. 2, 1940, bronchoscopy was performed to determine the source of positive sputum. There were productive changes in the right upper lobe, but no drainage. The left side revealed many islands of granulation tissue and considerable discharge. Pneumothorax was recommended on the left side. Nov. 30, 1940, pneumothorax was induced on the left, with conversion of sputum in one month. Sputum was still negative at the time of discharge, March 30, 1941. (Fig. 2.)

Case 3.—C. B. was a white man, 37 years old, whose onset occurred in 1919. The first positive sputum was found in May, 1937. Right pneumothorax was induced. Jacobaeus operation was performed in Dec., 1937. Sputum was negative one year; Aug., 1939, spread to the left upper lobe. Left pneumothorax was induced. Good anatomic collapse was achieved. Sputum remained positive. Bronchoscopy performed in July, 1940, to determine the source of positive sputum showed chronic inflammatory reaction and purulent discharge in the right upper

lobe branch. The left upper lobe branch appeared normal. Discontinuance of right pneumothorax and replacement by thoracoplasty were recommended. (Fig. 2.)

CASE 4.—J. T., a white youth, 18 years of age, had had a bilateral caseous pneumonic lesion two years previously. Sputum was positive. April, 1938, pneumothorax was induced on right. Sputum continued positive. May, 1939, pneumothorax was induced on the left. Sputum was still positive. Bronchoscopy was performed Dec., 1939, to determine the source of positive sputum. Granulomatous reaction was found in the left upper lobe branch. Jacobaeus operation, on the left, was recommended. Feb., 1940, Jacobaeus operation was performed. Sputum converted June, 1940. (Fig. 2.)

CASE 5.—M. L., a white woman, 34 years old, suffered from pleurisy with effusion on the left, in 1933. In 1937, an exudative lesion appeared in the left upper lobe. Pneumothorax was unsuccessful. Nov., 1939, there was spread in the left upper lobe, minimal infiltration in the right apex, and hemoptysis. Dec., 1939, two-stage thoracoplasty was performed. Cough continued but sputum was negative to May, 1941, then positive. Pain was felt in the right side and râles were heard in the right axilla. X-ray showed spread in the right upper lobe. Tomographs failed to show cavity on the left side. Bronchoscopy, Aug., 1941, showed infiltrative lesion in the left upper lobe; drainage was positive for tubercle bacilli. No change occurred in the mucous membrane of the right upper lobe. March, 1942, revision thoracoplasty was performed on the left. Sputum was negative. Cough disappeared, but returned after "cold" with atelectasis of the left lower lobe. (Fig. 3.)

CASE 6.—R. D., was a white man, 30 years old, whose onset occurred in May, 1937, with hemoptysis. Fibrocaseous lesion was found in the right upper lobe; exudative and productive, left upper lobe. Sputum was positive. After one year of sanatorium care, the patient was discharged as arrested, June, 1938. Oct., 1939, reactivation with positive sputum occurred. Nov., 1939, pneumothorax was induced, with fair collapse. Sputum was alternately negative and positive to March, 1941. Bronchoscopy, May, 1941, was repeated in Sept., 1941, and again in Feb., 1942, for source of positive sputum. Right upper lobe showed productive changes in mucous membrane indicative of healing. No change in therapy was recommended by bronchoscopy. There was an exudative reaction on the left side. Oct., 1941, pneumothorax started on the left side. Sputum remained positive at long intervals. Clinical progress was good. (Fig. 4.)

CASE 7.—E. T. was a white girl, 16 years old, whose onset occurred March, 1940. There was an exudative lesion in the axillary angle of the right upper lobe. Pneumothorax was induced on the right. Sputum was positive by culture only. Bronchoscopy in March, 1940, showed edematous masses partially obstructing the right upper lobe branch. Bronchoscopy in March, 1941, showed the exudative lesion subsiding. Sputum was negative. (Fig. 5.)

CASE 8.—J. Z. was a white woman, 22 years old. Her condition had been diagnosed by routine school x-ray in 1935. Sputum was negative. Dec., 1936 to May, 1937, the patient was given sanatorium care. In June, 1940, sputum was positive. The patient was admitted to the hospital in Sept., 1940. Pneumothorax was started on the right in Sept., 1940. Nov., 1940, Jacobaeus operation was performed. Anatomic collapse was good, but the cavity remained open. March, 1941, bronchoscopy showed an edematous nodule in the right upper lobe producing valve mechanism and tension cavity. (Fig. 6.)

CASE 9.—M. W., a white woman, 25 years old, had suffered one year's cough, loss of weight, two months' sweats, chills, and fever of 102 to 104° F.; there

was a wheeze on the right side. X-ray, April 23, 1941, showed extensive involvement of the hilus glands. Sputum was positive. Pneumothorax was contraindicated. June 3, 1941, bronchoscopy demonstrated ulceration of the trachea and right bronchus, with impaired drainage. Repeat bronchoscopies were performed July 10 and Aug. 14, 1941. Cough was reduced and temperature fell gradually to normal. Sept. 2, 1941, the patient complained of pain in the right side and dyspnea. X-ray picture Sept. 5, 1941, revealed spontaneous pneumothorax on the right side. Pleural pressure was minus 3, plus 4. There was massive caseation in the right lower lobe. The patient died Sept. 10, 1941.

CASE 10.—A. D. was a white woman, 20 years old, whose contact was her mother, an open case. There was partial atelectasis of the left upper lobe. Bronchoscopy showed external pressure on the posterior wall of the left stem bronchus at the upper lobe branch. Sputum never became positive.

CASE 11.—C. B., a colored woman, 21 years old, was operated upon for tuberculous cervical lymph adenitis in Aug., 1941. Roentgenogram showed large mediastinal nodes on the left. Sputum was negative. The patient was admitted to the hospital in Nov., 1941. A large mass was found near the left lung root and there was diminished aeration of the left lung. Bronchoscopy was performed Nov. 22, 1941. The left stem bronchus was encroached upon by external pressure 1 cm. below bifurcation. There was no perforation. Sputum remained negative. June, 1942, repeat bronchoscopy showed perforation inside the mouth of the left upper lobe branch. Sputum was positive.

CASE 12.—N. B. was a colored man, 23 years old, whose onset occurred in Aug., 1940; symptoms were fatigue, cough and wheeze, and loss of weight of seventeen pounds. April, 1941, an x-ray diagnosis was made of right tuberculous hilar lymphadenopathy. Sputum was negative. The patient was admitted to the sanatorium in Sept., 1941. Bronchoscopy was performed Oct., 1941; a granulomatous mass was found above mouth of upper lobe bronchus. On pressure, cheesy material was exuded. One culture was positive. Gradual absorption of the hilar mass was shown by x-ray film. The patient was discharged in June, 1942. Sputum was negative.

CASE 13.—K. M. was a colored man, 20 years old. His condition was diagnosed Nov., 1940, by x-ray survey. Sputum was negative. Jan., 1941, the patient was admitted to the sanatorium for preventorium care. In Feb., 1941, gastric lavage was positive for tubercle bacilli. Sputum continued Gaffky II. Aug., 1941, bronchoscopy showed a perforated extrabronchial lymph nodule near the mouth of the right upper lobe branch exuding caseous material. Re-examination in Nov., 1941, showed a granulomatous wart, without caseous discharge. (Fig. 7.) Healing was complete at examination in June, 1942.

CASE 14.—A. H., a colored man, 39 years old, had been under treatment for syphilis at the Board of Health clinic. March, 1938, routine x-ray pictures showed suspicions of tuberculosis. Sputum was positive, occasionally. Feb. 2, 1939, he was admitted to the sanatorium; sputum was positive to July, 1939, negative to Nov., 1939, positive Dec., 1939, then persistently negative. The source was undetermined. Bronchoscopy Jan. 27, 1940, showed healed perforated hilus gland (Fig. 8). The patient was discharged June 18, 1940.

CASE 15.—G. K., a white woman, 43 years old, whose onset occurred May, 1939, had suffered cough and weight loss, but sputum was negative. She was hospitalized Aug., 1939. There was occasional positive sputum. Right pneumothorax was induced Jan., 1940. Jacobaeus operation was performed May, 1940. This was followed by conversion of sputum. June to Oct., 1940, there was an occasional

low Gaffky count. Bronchoscopy was performed Nov., 1940, for source of bacilli. An enlarged gland was found extruding caseous material through toothpick-sized openings above and below the right middle lobe carina (Fig. 9).

CASE 16.—E. L., a white woman, 38 years old, had had a hilus gland infiltration and minimal parenchymal lesion on the left side for four years. Sputum was positive and there was segmental atelectasis of the lower lobe anteriorly. Bronchoscopy, March, 1940, demonstrated a granulomatous mass occluding the medial branch of the left lower lobe. (Fig. 10.)

CASE 17.—M. I., a white woman, 21 years old, had suffered from pleurisy one year previous to admission on Oct. 4, 1933. She complained of chill, pain on the left side, and headache. X-ray films showed atelectasis of the left lower lobe. Sputum was found negative nine times. Bronchoscopy was performed Nov. 12, 1933. The left stem bronchus was occluded by a cyanotic sloughing mass. Biopsy showed lymph tissue with many tubercles (Figs. 10 and 11). After bronchoscopy, sputum was persistently positive.

CASE 18.—G. H. was a white woman, 33 years old. April, 1938, enlargement of hilus glands was found. There was also a questionable lesion left parenchyma. Pneumothorax of the left side was followed by development of wheeze. Bronchoscopy was performed in April, 1940. There was collapse of the bronchial wall of the left lower lobe. Sputum was low Gaffky. There was a contact ulcer due to pressure between ulcerated surfaces (Fig. 12).

CASE 19.—R. B., a white woman, 43 years old, had a caseous pneumonic lesion of the left upper lobe for two and one-half years. Sputum was positive. There was pneumothorax of the left side and atelectasis of the left lung. Bronchoscopy

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Fig. 1 (Case 1).—Acute tuberculous bronchitis, showing injected pallor of bronchial mucous membrane, with moderate degree of redness and edema at mouth of draining bronchus.

Fig. 2 (Cases 2, 3, and 4).—Chronic tuberculous bronchitis (granulomatous variety) showing generalized redness and thickening of mucous membrane. In the draining bronchus are demonstrated irregularity of carina, granulomatous mammilations, and discharge of a milky liquid, positive for tubercle bacilli.

Note: Each illustration is intended to represent a type lesion which may be found in any bronchus. Hence, Fig. 2 is used for Cases 2, 3 and 4, although the pathology is in the left upper lobe in one case and the right upper lobe in another.

Fig. 3 (Case 5).—Chronic tuberculous bronchitis (infiltrative variety) showing indurative thickening of mucous membrane of draining bronchus and carina, downward displacement and cyanosis of the upper lobe branch following thoracoplasty, and increased mucopurulent discharge positive for tubercle bacilli.

Fig. 4 (Case 6).—Chronic tuberculous bronchitis (productive variety) showing thin carina, mucous membrane pale pinkish-yellow in draining bronchus; cartilaginous rings are visible. There is little or no discharge; generally negative for tubercle bacilli.

Fig. 5 (Case 7).—"Allergic" tuberculous bronchitis. Mucous membrane is pale, draining bronchus occupied by large, irregular elevations, the dominant character of which is edema. There is little or no discharge, generally negative for tubercle bacilli.

Fig. 6 (Case 8).—Check-valve mechanism; edematous mass in chronic inflammatory field obstructs partially stenosed bronchus, permitting ingress, but checking egress of tidal air.

Fig. 7 (Case 13).—Granulomatous wart in healing of perforated lymph gland; perforation first seen three months previously. Healing complete seven months later.

Fig. 8 (Case 14).—Crater of healed perforated lymph gland showing floor of yellow scar tissue and substitute mucous membrane. Anthracotic deposits are visible in the remnant of a gland.

Fig. 9 (Case 15).—Caseating lymph node at carina of right middle lobe branch, showing perforations into both middle and lower lobes. Pressure caused exudation of cheesy material.

Fig. 10 (Case 16).—Intrabronchial mass composed of granulation tissue and debris from perforated glandular and bronchial wall.

Fig. 12 (Case 18).—Cyanosis and congestion of mucous membrane, showing flattening of lumen due to destruction of cartilage, transference of lesion to opposite side by direct contact; bread and butter surface due to separation by descending bronchoscope.

Drawings by Milton S. Lloyd

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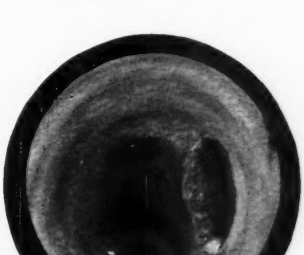

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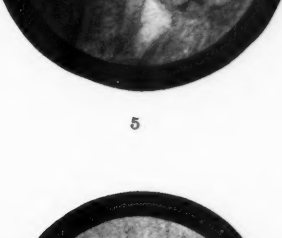

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was performed Jan., 1940, and extensive ulceration of the left stem bronchus in the mouth of the upper lobe branch was found (Fig. 13). The subsequent course was unfavorable.

Fig. 11 (Case 17).—Biopsy from left stem bronchus showing segment of lymph gland, including several conglomerate tubercles.

CASE 20.—B. D., a white woman, 32 years old, was seen with the onset in May, 1938. She was admitted to the hospital in March, 1940. Sputum was positive, and wheeze was present. Bronchoscopy, April 2, 1940, demonstrated ulceration of the right wall of the trachea with islands of healthy mucosa, and of entire right stem bronchus. Relief was afforded by removal of false membrane and retained secretions. April 8, 1940, right pneumothorax was induced. July, 1940, Jacobaeus operation was performed, followed by increased wheeze and dyspnea. Aug., 1940, emergency bronchoscopy afforded relief by suction removal of sloughing membrane and retained secretions. *Monilia nigrans* was cultured. Partial re-expansion of pneumothorax to improve drainage was advised. Oct., 1940, third bronchoscopy revealed stenosis trachea; there was a pinpoint opening in the right stem bronchus (Fig. 14) which still drained but no evidence of exchange of air. In Nov., 1940, at the time of the fourth bronchoscopy, the stenosed trachea would not admit a 7 mm. bronchoscope.

CASE 21.—B. B. was a colored woman, 34 years old, in whom the onset with hemoptysis occurred July, 1936. X-ray picture showed a far-advanced lesion with cavity on the right. Sputum was positive and pneumothorax was unsuccessful.

Phrenic nerve was resected June, 1937. Three-stage thoracoplasty was performed in March and April, 1938. The patient was discharged as arrested Sept., 1939. In April, 1941, positive sputum recurred. Bronchoscopy Sept., 1941, for source of positive sputum, revealed cicatricial closure of the right upper lobe bronchus with a pinpoint opening from which caseous and liquefied material continually exuded (Fig. 15).

CASE 22.—E. G., a white woman, 39 years old, whose onset occurred in 1933, had multiple cavities in the left upper lobe. In Feb., 1934, sputum was positive. Pneumothorax on the left was unsuccessful. Thoracoplasty was advised but never carried out. There was a gradual improvement until July, 1938, when sputum became negative. It has been persistently negative since. Bronchoscopy, Dec., 1939, showed the left stem bronchus closed by a healthy fibrous tissue floor (Fig. 16). The patient was discharged with the condition arrested July, 1940.

CASE 23.—M. H., a white man, 30 years old had a right upper lobe cavity. There was complete pneumothorax for one year with supervening atelectasis in the collapsed lung. Bronchoscopy showed distortion characteristic of right pneumothorax. Obstruction was completed in the right stem bronchus by a mucous plug (Fig. 17).

CASE 24.—J. C. was a white woman, 32 years old, whose onset occurred Nov., 1935. Sputum was positive. Sporadic treatment was given until March, 1940. When the patient entered the hospital wheeze was noticed. There was extensive involvement of the left upper lobe. Pneumothorax was begun on the left side, June, 1940. Jacobaeus operation was performed Oct., 1940. There was atelectasis of the left lung; March, 1941, bronchoscopy showed torsion of the left stem bronchus with the upper lobe branch below the level of the lower lobe branch (Fig. 18).

Fig. 13 (Case 19).—Cyanosis and thickening of mucous membrane, showing upper lobe carina displaced medially by collapse. Lower lobe is flooded by mucopus containing some air. Ulcer extends upward from upper lobe branch covered by false membrane, removal of which causes bleeding.

Fig. 14 (Case 20).—Lower end of trachea after removal by suction of large masses of false membrane, some of which can still be seen clinging to wall; considerable intact membrane remains. Right stem bronchus is completely ringed by cicatricial tissue but still remains open for drainage and still aerates lung.

Fig. 15 (Case 21).—Right upper lobe bronchus reduced to pinhead size by firm cicatricial tissue after thoracoplasty. Necessary lumen is maintained by continuous drainage; patient is fever free. (Compare with Case 31.)

Fig. 16 (Case 22).—Left stem bronchus closed by firm cicatricial floor; patient symptom free.

Fig. 17 (Case 23).—Deformity of the carina characteristic of pneumothorax of the right side. There is clockwise rotation and inbulging of the floor and lateral wall of the right stem bronchus, which is occluded by undrained discharge.

Fig. 18 (Case 24).—Tortion of left stem bronchus. The upper lobe branch is dragged down below the level of the lower lobe branch. Drainage has failed but the field has been cleared by suction.

Fig. 19 (Case 25).—Right upper lobe bronchus brought into view by the alignment effect of pneumothorax. Although the lobe had been totally airless and sputum negative for over two years, no closure of any bronchi up to third division branches could be seen.

Fig. 20 (Case 26).—Right upper lobe bronchus following thoracoplasty. The roof has been buckled in and the cartilage (apparently fractured) is seen within the lumen. This, however, failed to close the bronchus which continues to discharge brownish-gray pus.

Fig. 21 (Cases 27 and 28).—Carina between lower lobe branches in collapsed atelectatic lung; marked cyanosis and swelling. This unhealthy mucous membrane is likely to break down at the mouth of the draining bronchus.

Fig. 22 (Case 30).—Stricture of bronchus composed of fibrous tissue and covered by flattened mucous membrane. At first thought to be congenital, dilatation and biopsy were followed by positive sputum and homolateral spread. A plug of gray inspissated mucus is shown.

Fig. 23.—Normal view for comparison, showing velvety pinkish-red mucous membrane, knife-edge carina, visible cartilaginous rings, absence of secretions, and open bronchial lumena. Fatty nodule shown here is rarely seen.

Drawings by Milton S. Lloyd

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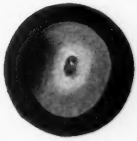

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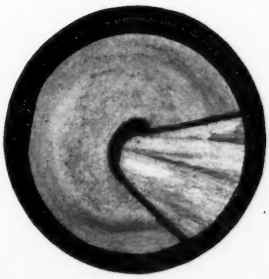

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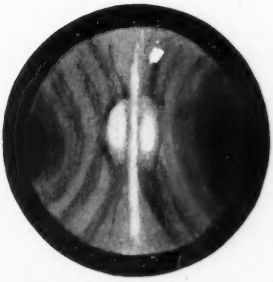

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CASE 25.—D. B., a white girl, 17 years old, had suffered from caseous pneumonic tuberculosis of the right upper lobe. There had been pneumothorax for three years. Jacobaeus operation had been performed. Sputum had been negative two and one-half years. Bronchoscopy done to determine the condition of the right upper lobe bronchus showed all branches open. Mucous membrane was intact (Fig. 19).

CASE 26.—W. S., a white man, 34 years old, on May, 1939, had an onset with hemoptysis. There was far-advanced bilateral caseous pneumonic tuberculosis and sputum was positive. Jan., 1940, there was pneumothorax on the left side, with conversion of sputum, which in April, 1940, spread on the right side. Sputum again was positive. Sept., 1940, pneumothorax of the right side was unsuccessful. Feb. to May, 1941, three-stage thoracoplasty was performed on the right side. Sputum was still positive. Jan., 1942, bronchoscopy showed granulomatous and edematous reaction in the left upper lobe, with fracture of cartilage and healing ulcer in the right upper lobe (Fig. 20).

CASE 27.—H. N. was a white woman, 35 years old, whose contact was her husband, 1931. X-ray showed left upper lobe lesion. The patient was first hospitalized Dec., 1937. Sputum was positive. Pneumothorax was induced on the left side, Nov., 1939. Jacobaeus operation was performed Feb., 1940. The patient developed wheeze after operation, and the lung quickly became airless. Sputum was Gaffky IV to IX. Bronchoscopy was performed June, 1940; the lung was flooded by retained secretions. Cyanosis and edema of the mucous membrane were found. This was considered a preulcerative stage (Fig. 21). Partial re-expansion to improve drainage was recommended. Repeat bronchoscopy was performed Oct., 1940. The lung was still flooded and there was ulceration at the mouth of the left upper lobe. Re-expansion of the lung was permitted to secure a selective collapse. Immediate improvement of patient followed. Sputum was negative and ulcer healed, Nov., 1941.

CASE 28.—M. F., a white woman, 25 years old, had "pneumonia" in the left side in July, 1938. Aug., 1938, sputum was positive. Sept., 1938, pneumothorax of the left side was induced. Dec., 1938, sputum was negative but condition was poor. The patient was admitted to the sanatorium in Feb., 1939; sputum was positive for four months. June 13, 1939, bronchoscopy showed atelectasis, cyanosis, and congestion of the left lower lobe. Sputum was negative for seven months, but her condition continued to be poor. Feb., 1940, sputum again became positive. April 10, 1940, bronchoscopy showed ulcer at the mouth of the left upper lobe. June, 1940, sputum was negative and continued to be so until Oct., 1940, when it turned positive again with the appearance of a large cavity on the right side.

CASE 29.—M. C., was a white woman, 20 years old. The onset occurred May, 1941. There was a slight infiltration of the right upper lobe; sputum was negative. May, 1942, the lesion showed progress. Sputum became positive in June, 1942. Bronchoscopy in Aug., 1942, showed an ulcerative lesion extending from the right upper lobe bronchus along the lateral wall upward for about three-quarters of an inch. Pneumothorax was recommended on the right side. Repeat bronchoscopy two months later showed the ulcer healed with partial stenosis of the right upper lobe bronchus. This branch was rotated backward by pneumothorax and drainage was much impeded. It was recommended that the patient lie face downward for specified periods. This was done with immediate improvement in drainage. Sputum was negative in Dec., 1942.

CASE 30.—L. W., a white woman, 28 years old, "always" had a wheeze on the left side. She had suffered ten years from repeated attacks of cough and fever.

Sputum was negative and there was atelectasis of the left lung. Bronchoscopy showed cicatricial stenosis in the left stem branch (Fig. 22). After dilatation (ten treatments) sputum became positive with homolateral spread.

CASE 31.—H. M. was a white woman, 29 years old, whose condition was diagnosed, April, 1938, as bilateral caseous pneumonic lesion and giant cavity on the left side. Sputum was Gaffky VIII. Pneumothorax was unsuccessful. Bronchoscopy showed extensive ulceration of the left lower lobe and partial obstruction by granulations and false membrane. Phrenic crush was performed Oct., 1939, with no effect. Monaldi's drainage was performed Aug., 1940. Sputum became negative two weeks later. The cavity was closed and the tube removed in Nov., 1940. The patient was then free of cough or fever; however, the cavity reappeared early in 1941 and cavernostomy was done in June. Again, the cavity closed and by April, 1942, the sinus had healed and the patient was symptom free; this was classified as an arrested case and the patient was sent to a sanatorium for convalescence. In June, temperature began to rise and in two weeks reached 104° F. Bronchoscopy showed ulcer in the left lower lobe healed and lumen closed by firm scar tissue. There was an acute inflammatory reaction and stagnant secretion in the closed branch. Immediate re-establishment of external drainage was recommended.

SUMMARY AND CONCLUSIONS

1. Tuberculosis in the parenchyma of the lung is always accompanied by changes in the mucous membrane of the draining bronchus. These changes are of the same nature as the pulmonary lesion and vary with the progress of that lesion.

2. They may be studied bronchoscopically in the visible airways. This method of examining the lung, therefore, serves as a most important guide to the management of collapse therapy.

3. An attempt is made to classify tuberculous lesions of the bronchi so that they and their significance may be more easily recognized. To this end, color drawings of the various lesions, together with the reports of the cases from which they are taken, are included.

4. Bronchoscopy emphasizes, from a new angle, the dangers inherent in artificial pneumothorax. It demonstrates clearly the necessity of interfering as little as possible with the drainage and ventilation of the uninvolved lobes and lung. It provides additional support to the belief that every pneumothorax should be a selective pneumothorax and that a contraselective pneumothorax may be actually harmful. At the same time, it provides an additional means of knowing when pneumothorax has failed and when it should be discontinued or changed in its mode of application or replaced by another method.

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MASSIVE CONGLOMERATE LESIONS OF SILICOSIS DIFFERENTIATED FROM PULMONARY NEOPLASM

RICHARD A. RENDICH, M.D., AND MORTIMER RICHARD CAMIEL, M.D.
BROOKLYN, N. Y.

MUCH excellent work has been done in defining the roentgenologic picture of silicosis. In spite of this, however, there are not yet any pathognomonic criteria which can be applied to the diagnosis of the atypical individual case. The massive conglomerate silicotic lesions, especially, present a difficult problem. Gardner[6] and others have noted their perplexing nature, particularly when confirmatory evidence, such as generalized nodulation, is absent. The confusion of this type of silicosis with the various inflammatory entities has been stressed in the past. Occasionally, however, the diagnosis of neoplasm is mistakenly made. For surgical, prognostic, medicolegal purposes, the need for exact roentgenologic interpretation is apparent.

Bradshaw and Chodoff[2] describe the case of a 34-year-old man on whom a pneumonectomy was performed under the impression that he was suffering from a pulmonary neoplasm. Although there was a history of the patient being exposed to coal dust, he presented a clinical and roentgenologic picture of neoplasm. An enlarged right hilar shadow was noted. There was no evidence of nodular lesions elsewhere in the lungs. At operation, a diagnosis of pulmonary carcinoma was made and a total pneumonectomy performed. Pathologically, the lesion proved to be anthrosilicosis. Pohle and Ritchie[13] recently reported an instance of silicosis and tuberculosis in which the pulmonary picture so strongly resembled a neoplasm that the patient was given deep roentgen therapy. The patient was a 36-year-old man who gave no history of exposure to silica. His chief complaint was dyspnea. Roentgenographic study showed a large mass extending outward from the right hilus, and another slightly smaller projecting from the opposite hilus. Therapy was administered to the supposed neoplasm but autopsy proved the disease to be silicosis with tuberculosis. Gut[8] regarded a roentgen shadow in one lung to be a primary carcinoma. He interpreted a shadow in the opposite lung to be metastatic. An autopsy disclosed anthracotic induration at both hila. Blanco and Dighiero[1] resorted to pulmonary punch biopsy in a suspected case of neoplasm. The patient was found to have silicosis. Gardner[6] states that the conglomerate lesions have been mistaken for tumors, healed infections, and pulmonary atelectasis.

From the Department of Roentgenology of the Kings County Hospital, Brooklyn.
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Garland[7] likewise notes that silicosis may be mistaken for carcinoma. Holman and Pierson[9] report the case of a 58-year-old man who had been a quartz miner for twenty-eight years. A consolidation in the right lower lung field was interpreted as probably being due to neoplasm. Pneumoconiosis was only secondarily considered. Autopsy revealed the patient to have had pneumoconiosis and tuberculosis. Conversely, Pendergrass[12] reminds us that the infiltrating or permeating malignant metastases may be of such appearance as to resemble pneumoconiosis of a conglomerate type.

It will be seen, therefore, that in cases of conglomerate silicosis, a mistaken diagnosis of pulmonary neoplasm is probably made more often than is suspected. We are presenting two cases of pneumoconiosis with which we had a similar experience.

CASE HISTORIES

Case 1.—J. H., a 73-year-old white man, was admitted to the Kings County Hospital on Feb. 11, 1942. He had no known occupation. He was too ill to give an adequate history. His chief complaint apparently started three months before, when he fell down a flight of stairs and fractured several of his left ribs. Since that time he complained of a cough which was occasionally bloodstreaked. In addition, there was weight loss of unknown amount and marked weakness. These complaints continued until the time of admission. Additional history could not be obtained.

Physical examination showed a chronically ill, emaciated, white man with rapid respiration but no marked distress. No cyanosis was present. His mentality appeared dulled. Examination of the head and neck was negative. The trachea was in the midline. The neck vessels were not distended. No cervical glands could be palpated. Examination of the chest revealed a symmetrical thorax. The heart was not enlarged. The blood pressure was 130/80. The pulse was of a regular sinus rhythm with a rate of 100. There was dullness over both lung fields, especially the right, in the axillary region, and over the lower portion, posteriorly. There was flatness at the right base. Breath sounds were diminished and there were fine and medium râles heard bilaterally. Examination of the abdomen was essentially noncontributory. Some small bilateral inguinal glands were palpated. Both feet showed a 2 plus edema. The reflexes were normal.

Laboratory studies revealed a negative Wassermann reaction; urine was negative. The blood count showed 9200 W.B.C. with 93 per cent polymorphonuclear leucocytes. Hemoglobin was 70 per cent; urea was 26. The temperature varied between 99 and 100° F. with a terminal rise to 101° F. The patient did not expectorate and no sputa could be examined. A gastric washing was positive for acid-fast bacilli.

Roentgen study of the chest (Fig. 1) revealed a large dense consolidation in the right lower lung field apparently extending outward from the hilus. A few cherry-sized nodulations were noted above the main consolidation. In the opposite central lung field was another consolidation, of similar density to that found on the right, with an almost clear zone around its circumference. Both apices were clear. The original roentgenologic report read, "There is evidence of a bilateral massive consolidation in both central lung fields with slight displacement of the trachea to the right, rather due to shrinkage of inflammatory condition than bronchogenic new growth." However, the roentgenogram was presented at conference to the entire roentgenologic staff and the concensus of opinion was that the patient was suffering from a pulmonary neoplasm on the right, with metastases to the left.

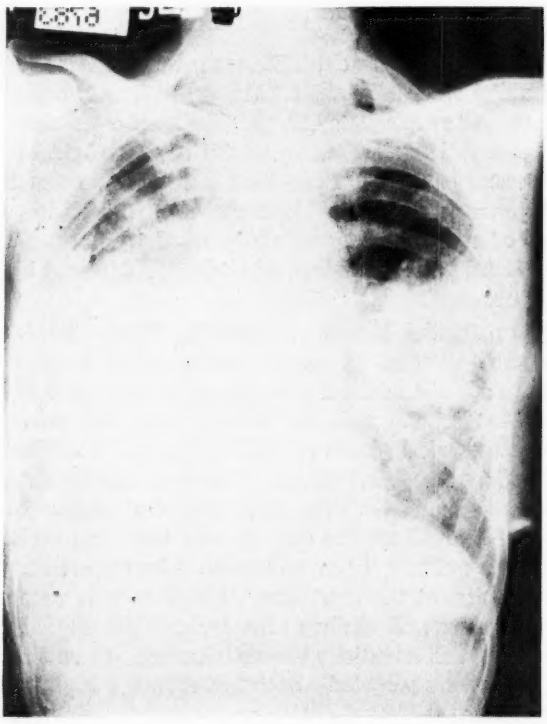

Fig. 1 (Case 1).—Massive conglomerate silicotic lesion mistaken for pulmonary neoplasm with metastases.

The patient became progressively weaker and died on Feb. 25, 1942, fourteen days after admission. The final clinical and roentgenologic diagnosis was carcinoma of the right lung with metastases to the opposite side.

Autopsy.—The lungs were found to contain a tremendous amount of anthracotic pigment giving their surfaces the appearance of black patent leather. The right lung weighed 850 Gm., the left 800 Gm. The lower and middle lobes on the right were diffusely hard. On section, these were black and gray in color and resembled glistening marble. Some areas were soft and a puslike substance could be expressed. Numerous tubercle-like structures were found near the hilus. The upper lobe on the right had a doughy feel and its cut surface was black. On the left, the base of the upper lobe and the upper portion of the lower lobe were particularly involved. A diffuse mass was found here which was mottled with gray and black areas. Tubercle-like structures also could be made out. The borders of the lung were emphysematous, especially the apex of the upper lobe. In addition, tubercles were found in the kidneys, spleen, and liver.

The final pathologic diagnosis was pneumoconiosis; bilateral silicotuberculosis, tuberculosis of the kidney, spleen, and liver.

Subsequent occupational history was obtained from the New York City Department of Public Welfare, from which the patient had received relief. The occupational history revealed that the patient had been a coal miner from 1893 to 1918.

Case 2.—A. T., a 45-year-old Negro, was admitted to the Kings County Hospital on May 16, 1941. He had been x-rayed at another hospital and referred as a case of carcinoma of the lung. His occupation was given as that of an iron worker, duration not noted. He had been fairly well until two weeks before admission when he caught a cold and had a sore throat. He had lost ten pounds in the previous eight months. He had no chest pain, hemoptysis, or night sweats. A productive cough with whitish sputum was present for unknown duration. His past history was noncontributory with the exception that he had had recurrent attacks of nocturnal dyspnea for several years and occasional ankle edema. No history of previous surgical or medical illness was obtained. Venereal disease was denied.

Physical examination revealed a mentally dulled, well-developed and well-nourished man, who appeared chronically ill. His blood pressure was 138/82. The positive physical findings were diffuse scattered wheezes and squeaks throughout both lung fields with dullness over both upper fields. The liver was down one finger below the costal margin.

Laboratory studies revealed the sputa to be negative for tubercle bacilli four times. The Wassermann reaction was 4 plus. The electrocardiogram showed myocardial fibrosis. Urine was negative and blood count showed 7300 W.B.C. and 2.8 M. R.B.C. with 48 per cent hemoglobin.

Roentgen study of the chest (Fig. 2) was reported as follows: "Evidence of a moderate sized irregularly nodular-like area of infiltration extending from the roots of the right lung to the periphery of the hemothorax on this side, confined mainly to the mesial two-thirds of the right upper lobe. A similar such nodular-like infiltrative process is also noted extending from the superior plane of the root of the left lung to the left subclavicular region. The roentgen impression is that of an

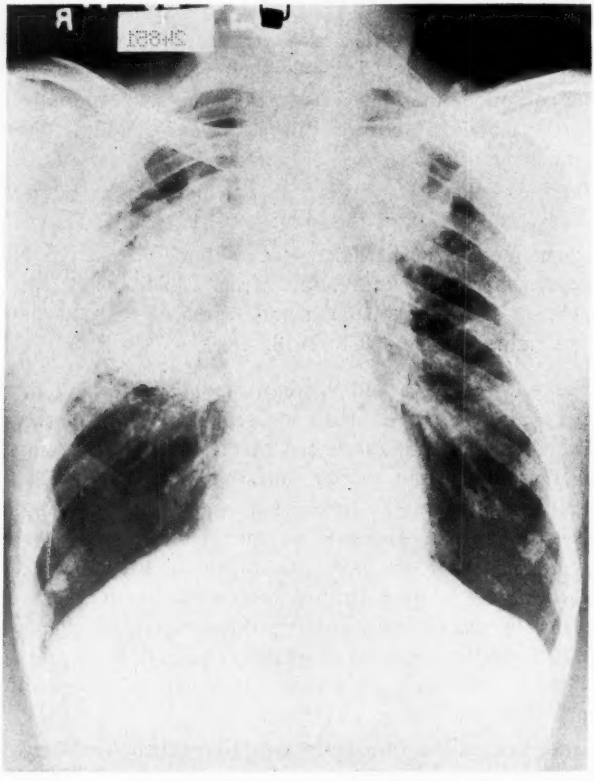

Fig. 2 (Case 2).—Massive conglomerate silicotic lesion mistaken for pulmonary neoplasm of lymphoblastic type.

underlying new growth on the order of lymphoma, however, one cannot entirely exclude the possibility of an underlying carcinomatous process. There is a small irregular nodular-like area of infiltration in the basal and peripheral portion of the right hemothorax and another such nodular focus somewhat larger in size noted in the peripheral aspect of the plane of the third right interspace." The roentgenogram was presented to the radiologic staff at conference and the concensus of

opinion was that the patient was suffering from a pulmonary neoplasm, most likely lymphoblastic in nature.

The clinical impression was that the patient was suffering from a bronchogenic carcinoma or lymphoblastoma and a therapeutic trial of the roentgen therapy was suggested, but therapy was deferred until the patient's clinical condition might improve under general supportive measures. However, his respiratory difficulty became more severe. His breathing was said to be that of a bronchiolar spasm and on June 13, 1942, he suffered an acute attack of dyspnea and died. The final clinical and roentgenologic diagnosis was lymphoblastic pulmonary new growth or bronchogenic carcinoma.

Autopsy.—The pleural surfaces of both lungs were purplish black in color. The cut surfaces of both lungs were black in color. The right lung weighed 600 Gm., the left 360 Gm. Both upper lobes were diffusely consolidated and the right contained a small encapsulated caseous area of the periphery. Anthracotic pigment could be scraped away in large quantities from the cut surfaces. The peribronchial nodes were average in size and filled with anthracotic pigment. The aorta showed a "tree bark surface." The microscopic picture confirmed the gross findings. The final pathologic diagnosis was pneumoconiosis; bilateral silicosis; syphilitic aortitis.

Subsequent occupational history obtained from the patient's sister and a friend revealed that he had been a coal miner and steel mill worker in Pennsylvania for an unknown period of years.

DISCUSSION

The massive conglomerate lesions of silicosis, as demonstrated in these cases, are usually due to the presence of associated infection.[6] Although in a great number of cases the tubercle bacillus is responsible, it is possible that other infections or no infection may be responsible. Pendergrass[12] gives an excellent résumé of the roentgen aspects of silicosis and asbestosis. While it may still appear impossible to differentiate between certain conglomerate silicotic lesions and neoplasm, nevertheless, an attempt to define their component characteristics may permit more exact diagnosis. Table I is a comparison of some of the differential points. While no single factor is decisive, the evidence in a particular case becomes cumulative and of sufficient weight to permit differentiation. The study does not include those rare cases in which both silicosis and neoplasm are found in the same patient.

The cases which result in the most confusion are similar to the first case presented. Usually there are two large consolidations, one in either lung field. Their size and density are such that at the same moment one is impressed by their neoplastic appearance and perplexed by their

TABLE I

DIFFERENTIATION BETWEEN THE MASSIVE CONGLOMERATE LESIONS OF SILICOSIS AND PULMONARY NEOPLASM

MASSIVE CONGLOMERATE SILICOSIS	NEOPLASM
1. Usually bilateral	1. Primary carcinoma is unilateral although metastases may be bilateral
2. Tendency toward symmetry	2. Asymmetrical, although the rare lymphogenic type tends toward symmetry
3. When lesions are small they may be asymmetrical; in such cases the tendency is to right-sided preponderance; if single, usually on right	3. Either side
4. Subapical or subclavicular location is common; usually located in the apices of the lower lobes	4. No favorite location
5. A clear zone is frequently found surrounding the lesion; this may be between the hilus and the lesion or the periphery and the lesion, or both	5. Main bronchus neoplasm is in relation with hilum; peripheral branch type is nodular parenchymal lesion separate from hilus
6. The shape is not likely to follow a standard pattern	6. Certain standard patterns are recognized
7. The density of the consolidation is usually greater than that of neoplasm	7. The density associated with atelectasis due to neoplasm is less dense than silicosis
8. Confirmatory typical nodulation may be present	8. Typical metastases may be present
9. Lesions in the opposite lung field incline toward the same density and general appearance as the side with the greater pathology; in some cases except for the size, the primary side could not be chosen	9. Metastatic lesions in the opposite lung field do not usually resemble the primary
10. With the accompanying active tuberculosis, an overexposed or Bucky-Potter film may reveal cavitation or calcification	10. Cavitation is rarely found, but when present is usually in a single large lesion
11. Emphysema of remaining lung fields is almost invariable	11. Emphysema infrequent
12. Fibrous strands may radiate from the lesion, frequently downward causing diaphragmatic deformities	12. Fibrosis is not prominent
13. Spontaneous pneumothorax is a rare complication, which may be seen with superadded tuberculosis	13. Unlikely
14. Interlobar and peripheral pleura are frequently thickened	14. Not present
15. Bronchography is indefinite	15. Bronchography may give typical appearance
16. Roentgen examination of other parts of the body is noncontributory except with the suggestive finding of tuberculosis	16. Roentgen examination of other parts may reveal definite metastases or a primary neoplasm
17. Radiation therapy does not favorably effect the lesion	17. Therapy may alter shape or size of lesion

TABLE I—CONT'D

MASSIVE CONGLOMERATE SILICOSIS	NEOPLASM
18. Occupational history is very significant (see Discussion)	18. Occupational history of dust exposure puts diagnosis of silicosis in favor
19. Almost invariably males	19. May be of either sex
20. Average age is under 50	20. Average age is over 50 (Craver[3])
21. May give long history	21. Average duration of complaints is 3.8 mo.
22. Dyspnea is more marked	22. Hemoptysis is more marked
23. Sputum may reveal dust	23. May reveal neoplastic cells
24. Positive sputum or gastric lavage for tuberculosis is suggestive; negative sputum does not exclude silicosis	24. Sputum usually negative
25. Bronchoscopy always negative for neoplasm	25. Settles the diagnosis when neoplasm invades a main bronchus
26. Biopsy taken during the bronchoscopy may give positive pneumoconiosis result	26. A positive biopsy for silicosis should militate against the diagnosis of neoplasm
27. Punch biopsy may give positive pneumoconiosis result	27. As No. 21; a positive test for neoplasm may be obtained
28. Thoracotomy may show typical appearance of pneumoconiosis; palpation of lung during exploration should be augmented by frozen section study, if possible, before pneumonectomy; at least one silicotic lung has been removed for neoplasm[3]	28. Thoracotomy may reveal metastases; the same comment applies in this column as in opposite
29. Physical examination of the other parts may be noncontributory	29. Physical examination may reveal primary neoplasm, metastases, glandular enlargements, etc., which would suggest diagnosis of malignancy

TABLE II

RELATIONSHIP BETWEEN SILICOSIS AND PULMONARY CARCINOMA FROM THE TABLE OF THE MINERS PHTHISIS MEDICAL BUREAU OF SOUTH AFRICA (1935)

	TOTAL AUTOPSIES	CARCINOMA OF LUNG	PER CENT OF ALL AUTOPSIES
European miners with silicosis	1438	10	0.70
European miners without silicosis	1629	12	0.71
European males never underground	1393	13	0.93

atypical form. In most instances described, and in our own, the examiners chose to call the larger of the conglomerations a primary carcinoma. Rationalization was then stretched to fit the picture and the opposite consolidations were called metastases. As seen from the diagnostic table, a bilateral lesion should have given pause to the diagnosis. Metastases from one side to the other could not be expected to behave in the manner noted. Metastases of such extent would be likely to reveal their identity by a more typical appearance. In these cases the opposite lung field contained real pulmonary infiltrations and consolidations which were smaller but of an identical nature with the opposite masses. A perusal of the table will show the following items to coincide with the case in question: 1 to 7, 9, and 24.

Our second case demonstrates the reasons for the inclination toward explaining the pulmonary picture as one of lymphoblastic origin. Here the similarity of the pathology in each lung field is more marked and the association with the hila more apparent. The peculiarity of our second case was in the appearance of a narrow but definite clear zone between the hila and the masses. The densities of the consolidations was far greater than could be expected from a lymphoblastic neoplasm. The nodules in the remainder of the lung field were extremely unusual for lymphoma. On re-examination a more or less typical silicotic nodulation can be made out in the left upper outer lung field. An emphysema of moderate degree is noted in the apparently uninvolved lung. Each of these might have given pause to the diagnosis of lymphoblastic neoplasm. Because of the atypical form the masses assumed, the examiners suggested carcinoma and tuberculosis as alternative diagnoses. A review of the diagnostic table shows the following points to fit the picture: 1 to 9, 11, and 20.

The diagnosis in those reported cases where the lesions were predominantly asymmetrical or unilateral or where the roentgen evidence was vague might be aided by the clinical points in the table. In the case of Bradshaw and Chodoff[2] an occupational history was elicited. In addition, a biopsy taken during bronchoscopy was interpreted as anthracosis. Many other diagnostic points fitted the picture. Likewise, the patient of Holman and Pierson[9] gave a twenty-eight year history of quartz mining.

The problem of simultaneous occurrence of silicosis and of malignancy, while not the principal subject of this discussion, merits some attention. While a positive occupational history should be strongly suggestive of the single diagnosis of pneumoconiosis, there are some who claim that the incidence of pulmonary carcinoma is higher among such patients. Klotz,[10] in an analysis of fifty autopsied cases of silicosis, found four instances of pulmonary carcinoma. This incidence of 8 per cent of pulmonary carcinoma in silicotics compared with 1.17 per cent in

4500 unselected autopsies. Lynch and Smith[11] report a case of asbestosis with pulmonary carcinoma. Their report is significant in that they believed they could demonstrate a metaplasia of the bronchial epithelium. They also raised the question of related etiology. Egbert and Geiger[5] and Desmeules and associates[4] likewise report cases of associated asbestosis and pulmonary carcinoma, and discuss a possible relationship. On the other hand, there have been some very authoritative investigations which conclude that there is no relationship. Vorwald and Karr[14] review the reports of eighteen authors who among them reported sixty cases of associated inhalation disease and pulmonary carcinoma. In addition, they investigated 178 autopsies of patients who had known occupational exposures. Of these, 136 were found to have silicosis. Of this number only 2 had pulmonary carcinoma (1.12 per cent) and 4 had carcinoma elsewhere. The authors published the table of the Miners Phthisis Medical Bureau of South Africa (1935) which revealed the figures found in Table II.

In addition, they cite the evidence against silicosis being the cause of the high incidence of pulmonary carcinoma among miners in Schneeburg and St. Joachemstal. The accepted opinion is that the miners inhale radioactive substances in addition to dust. They conclude, therefore, that there is no etiologic relationship between pneumoconiosis and carcinoma. The weight of statistical evidence appears to lie in their favor. Other experienced workers in the field, such as Pendergrass[12] are of the same opinion.

SUMMARY

1. The conglomerate lesions of silicosis may be mistaken for neoplasm.

2. For prognostic, medicolegal, and surgical purposes, the need for exact roentgenologic interpretation is apparent.

3. Two cases of the massive conglomerate type of silicosis which were mistaken for neoplasm are reported.

4. A table of differential characteristics between such lesions and neoplasm is presented.

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